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A MANUAL

OF THE

PRACTICE OF MEDICINE

BY

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ROYAL COLLEGE OF PHYSICIANS AND IN MATERIA MEDICA AND
PHARMACEUTICAL CHEMISTRY AT THE UNIVERSITY OF LONDON

FIFTH EDITION

LONDON
J. & A. CHURCHILL
7 GREAT MARLBOROUGH STREET
1898

L46
T24
1898

PREFACE TO FIFTH EDITION.

In preparing the fifth edition of this "Manual of the Practice of Medicine," the work has again been very carefully revised, some sections have been largely re-written, and notices of many diseases recently recognised or omitted from former editions have been added. Among the former are the introductions to Diseases of the Nervous System, and to Diseases of the Blood, and the subjects of Aphasia and Ringworm. Among the latter are Glandular Fever, Divers' Paralysis, Erythromelalgia, Angeioneurotic Œdema, Hypertrophic Pulmonary Osteo-arthropathy, and Tubercle of the Skin. A separate section is devoted to Diseases of the Mediastinum; and Filarial Disease and Hæmoglobinuria have been transferred to Diseases of the Lymphatic System and of the Blood respectively.

I have often in the text given my authority for statements made, but I may here mention that I have consulted many of the articles in Professor Clifford Allbutt's "System of Medicine," and that I am indebted to the "Twentieth Century Practice of Medicine," Manson's "Tropical Diseases," Coles' treatise on "The Blood," and many other works on special subjects.

It has been thought advisable to make a change in the style of the printing of the work, and it is hoped that the larger page, with a clearer type, will be found to be an improvement.

FREDERICK TAYLOR.

October, 1898.

involving a much wider area, but too obscure in their origin to demand a readjustment as yet.

By consulting the most recent works, especially those of Fagge, Strümpell, Payne, Ziegler, Gowers, M. Mackenzie, Douglas Powell, Ralfe, H. Morris, and Crocker, to whom I must express my indebtedness, I have sought to bring this book fully up to the modern state of knowledge. I have not, however, devoted much space to the discussion of theories, finding that the facts of medicine are amply sufficient to fill, and more than fill, a volume such as this, and being convinced that these facts require to be seized and held fast by the beginners in medicine, not only for the sake of diagnosis and treatment, but also for the right estimation of the various theories which are advanced. With a brief statement, therefore, of such views I have in most cases been content.

FREDERICK TAYLOR.

20, WIMPOLE STREET,
CAVENDISH SQUARE, W.
January, 1890.

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that each is the first essential lesion in the history of the patient's illness, although each owns some precedent cause, such as chill, or contagion by micro-organism in the case of pneumonia, alcoholic indulgence in the case of cirrhosis, and some hitherto unknown causation in the case of cancer of the stomach.

We also cannot deny the name of disease to the disturbances of anatomy that may follow such primary lesions in some cases. Thus, cirrhosis of the liver leads to effusion of liquid into the peritoneal cavity, abdominal dropsy or ascites; cancer of the stomach, involving the pylorus, causes secondary dilatation of the walls of the stomach; valvular disease of the heart causes congestion of the liver and kidneys, and dropsy of the feet and legs.

Then, again, as a result of either of these groups of anatomical changes, there are certain disturbances of functions, and abnormal subjective sensations, such as pain, vomiting, jaundice, the passage of albumen in the urine, and others. Are they also diseases? There is no difficulty in coming to a conclusion that in these also there is disease.

Lastly, we have to do with a class of disorders in which no structural change whatever can be found: these are mostly referable to the nervous system; they consist of pain, or spasm of muscle, or loss of sensation, or loss of power, or vascular changes secondary to functional alterations in the vasomotor nerves. This change in every case is for the time being disease, and the different instances are grouped as *functional diseases*.

But the subject may be approached from another point of view. Having decided that the term *disease* has a very comprehensive meaning, we may seek to determine how we shall employ the word *diseases* that is, how we shall arrange what we know of the possible changes of structure or function into separate groups for practical purposes. These practical purposes, in relation to diseases, are their recognition during life, when the investigation of structure cannot be so complete as after death, and their subsequent treatment. We then perceive that the various changes of structure which we cannot ourselves see are accompanied by certain other changes, secondary diseases, or functional disturbances, which are as many indications, or *symptoms*, of the deeper change. Thus, with pleurisy we have pain, cough, and shortness of breath; with cancer of the stomach, pain and vomiting. These indications may be subjective experiences on the part of the patient, like pain, [illegible] inability to eat or move; or objective signs to the [illegible], such as swelling, hardness, elevation of temperature. [illegible] [illegible] these objective signs must be mentioned especially [illegible] [illegible] known as *physical signs*, a term used mostly in [illegible] [illegible] examination of the chest and abdomen with the [illegible] [illegible] hand (palpation), and the ear (auscultation or percussion). Thus, *symptoms* and *physical signs* are regarded as forming the sum total of all the results of a structural lesion which can be manifest to the physician and guide him in his opinion.

Now, first, the patient has certain experiences of disorder and discomfort, which are to him a very real illness or disease; secondly, the physician, by his special knowledge, can show the existence of other disturbances unperceived by the patient; and thirdly, there are still facts which he may be unable to observe, and which are only revealed by the pathologist on *post-mortem* examination.

Are we to limit the term diseases to the last or to the second of this series, or may we allow it to be used also for the headaches, vomiting, dropsies, convulsions, &c., which are the most prominent part of the patient's suffering, but which are to the physician only the symptoms of something deeper, to which he is constantly striving to attain? Provisionally, no doubt, we must allow the term a wide application. For, in the first place, there is the whole class of functional disorders, which have, as far as our present knowledge extends, no anatomical basis, and which must, nevertheless, be distinguished from one another and treated; and secondly, there is the fact that lesions of the deeper organs may be very imperfectly revealed even by conditions which cause much suffering to the patient.

But though we may be sometimes compelled to think of dropsy, or headache, or neuralgia as a disease, we must remember that we should never rest content with that position until every effort has been made to find out what organ or structure may be at fault, and we should never permit ourselves to fall into the slovenly habit of ticketing all symptoms with the name of diseases, and treating them without any attempt to remove, or modify, the ultimate cause, when it may be with no great difficulty ascertained. This search after deeper causes, which is incumbent on every physician with regard to the case before him, is being prosecuted with the greatest vigour by the profession in the case of those disorders known as functional diseases; and it is possible that many of them will at no great distance of time be found to be due to structural changes, which at present elude our observation.

A thorough knowledge of every disease requires an acquaintance with several separate branches of study, which are as follows:—

Ætiology, the study of its causes in general. *Pathology*, the study of its causes within the body, and the processes resulting therefrom. *Morbid Anatomy*, the alterations in the structures caused by it. *Symptoms*, the indications—subjective on the part of the patient, and objective to the physician—by which we arrive at a knowledge of what is wrong. *Diagnosis*, the method of distinguishing from one another the diseases that may have symptoms

which act as predisposing causes at o
causes at another. *Ætiology* strictl
causation of disease, but it is perhaps
remoter causes, and to those condition
a disease, without its being known ho
rence. The relations to disease of age,
roundings, food, and preceding illnesses
under this heading. On the other han
the body immediately preceding or cau
sidered rather as pathological processes t

Pathology is sometimes used to sign
structures, but it is better limited to t
cesses—that is, practically, the influe
disease have upon the functions and stru
morbid anatomy or **pathological anatomy**
diseased structures themselves.

Symptomatology is the study of the sy
and associated with this and with its mor
consider what have been called **comp**
Complications are certain lesions or sy
result of the original disease, but only oc
and are not regarded as a necessary par
abscesses are a complication of enteric
common complication of phthisis; paroti
tion of pneumonia. But the term is applie
for instance, in enteric fever, rose spots
universally present, and yet they are r
disease and never as complications. We m
light the case where one disease occurs
but as far as our

symptoms, physical signs, or points in the history of the cases which, taken together, indicate that the patient is suffering from a particular ailment.

By *differential diagnosis*—a somewhat tautological term—is meant a knowledge, in any given disease, of all the other diseases which most clearly resemble it, and the points of difference upon which reliance may be placed to distinguish it. But the word *diagnosis* is not always used with the same extent of meaning. Some would limit its use to the cases where, after a careful consideration of the symptoms, signs, and history, aided by their knowledge of the pathological processes to which each organ is liable, they *infer* what they cannot actually see—namely, that the patient has this or that disease. But the term may be extended to the *recognition* of quite obvious lesions, where the amount of inference required is little or none, as, for instance, where one "diagnoses" a black eye, a lacerated wound, a fractured bone with the fragment projecting from the skin, or a pleural effusion after exploring the chest, and seeing the serum in the syringe. There is, however, no sharp line of distinction between these groups of cases, and they are all of them technically diagnoses. Similarly, it may be said they are all differential diagnoses, since the recognition of a particular disease cannot be considered sure unless the diseases most like it are deliberately, or instinctively, as it were, excluded. Every diagnosis requires obviously a careful consideration of the symptoms, physical signs, and other indications of disease; but, in addition, the former history of the patient and the duration of the symptoms are of the utmost importance; and finally, the physician must have a tolerably complete knowledge of the lesions to which each organ is liable, and of their relative frequency under different conditions. Probability is an important element in diagnosis. In exceptional instances a disease may present a physical sign, symptom, or indication which is not caused by any other known condition. Such a sign or symptom is said to be *pathognomonic* of the disease in question. As a fact, the number of *pathognomonic symptoms* is a very small one.

It will have been gathered from the remarks on diseases that by diagnosis we shall always aim at finding out the *primary* lesion; thus, we must not be content with calling any pain rheumatism or neuralgia, but we must try to find out whether such pain is caused by pressure on a nerve, by inflammation of a nerve, or by degeneration of nerve-tracts. But in many cases the patient suffers from numerous symptoms, pain, cough, sickness, dropsy, albuminuria, and others. As a rule, we should try to see how far all these conditions may be due to one single primary lesion, such as valvular disease of the heart, or granular disease of the kidney; but we must not forget that frequently two or more independent lesions coexist, and produce a complex arrangement of symptoms.

Prognosis.—Successful prognosis requires a thorough acquaintance with the natural history of every disease, with the extent to which the disease is influenced by age, sex, and other ætiological factors, and a careful judgment on the variations of the patient from day to day.

The questions that arise in prognosis are such as these:—Will the patient recover? Will he recover completely, or be left with any organ damaged? Will he ever have the disease again? If it is a fatal disease, how long will he live?

In the early days of a disease the question of recovery can only be answered by a consideration of the percentage mortality as known by statistics. As the case proceeds, the rapidity or severity of the symptoms, the condition of the circulation (pulse or heart), the ability to take food, the integrity of the nervous system—are the points which have most bearing on one's opinion. In practice, prognosis is often of the greatest importance for the physician's credit, and a hasty conclusion, which turns out wrong, is often remembered against him more than any want of success in treatment.

Treatment.—In this we should aim first at the removal of the cause where this is possible; if not, we may succeed in neutralising its influence. One or other of these methods may suffice to cure all the symptoms and troubles of the patients; but in most cases we are also called upon to deal directly with the symptoms, using remedies that have no influence upon the underlying disease. We must, when doing this, never forget that such symptoms hold a position secondary in importance to that of the disease which causes them. Lastly, we must in all cases counteract the tendency to death, which may, indeed, be the natural course of the disease, or may arise rather as an accident from some infrequent complication. As an example we may take phthisis, which is due primarily to the invasion of the tubercle bacillus. The removal of this, when once it has obtained a footing, cannot be directly effected. Its influence can be neutralised by the best hygienic surroundings, by fresh bracing air, and by special climates which enable the body to resist the action of the bacillus. In the meanwhile there are numerous symptoms—cough, expectoration, pain, sweating, diarrhœa—which will diminish as the condition of the [illegible] improves, and which can be also controlled by suitable [illegible]. In addition, serious complications may arise, especially [illegible], or spitting of blood, by which life is directly [illegible] and such a death may be averted by proper thera[illegible]. Pleuritic effusion is an instance in which we [illegible] [illegible], [illegible], pain, and distress due to the presence of [illegible], the removal of the liquid either by tapping, or by the use of drugs, is followed by relief of all the symptoms. One point it is very necessary to impress upon the student—namely, that diseases are to be treated not by medicines or surgical manipulations and operations alone, but by regulations as to rest or exercise, diet, hygienic surroundings, residence, and other considerations.

Prevention.—The *prevention* or *prophylaxis* of disease will only be occasionally referred to. It is almost co-extensive with sanitary science. It consists partly in avoiding the causes, partly in so preparing the body as to render it insusceptible to the operation of such causes: of this last method vaccination is an example.

CLASSIFICATION.

Some time ago diseases were divided into general and local, the latter being those in which particular organs or parts of the body were mainly or alone affected, such as the heart, the lungs, the brain, or the skin; and the former those in which the whole economy was disturbed, so that it could scarcely be said that one organ was more affected than another. The peculiarity of many of these complaints was that they were accompanied by fever, that they were contagious, or transmissible from one person to another, and that one attack protected the individual more or less completely against a subsequent attack.

Improved methods of research with the microscope led to proof that not only did the *contagium*, or *virus*, or medium of transmission from man to man, consist of visible particles, but that these particles were themselves living organisms, different in different diseases, capable of cultivation and reproduction both within and without the body. The study of these micro-organisms is the science of *Bacteriology*.

The general diseases were thus seen to be both infectious and specific, and we may instance typhus, scarlet fever, measles, small-pox, and influenza. But extended researches showed also that many diseases hitherto considered local were also due to specific and infective agents: for instance, pneumonia, tetanus, and diphtheria, as well as suppurative fever; and in their intimate pathology these disorders with their pronounced local manifestations must fall into line with the well-known fevers, such as typhus and small-pox. Classification then is rendered difficult by the fact that if the organs of the body are taken as a basis, there are diseases which affect the whole body simultaneously and no one organ in particular: but when these are investigated and their bond of union is found to be the infective micro-organisms, then it is seen that a number of diseases affecting mainly one organ or part of the body must be included with the infectious disorders; and as the tendency of all modern research is to discover microowever, an advantage in re diseases, formerly called local, as long as groupings, and it is this, that in their physic they are necessarily comparable with the oth the same group, whether the infectious na have been shown or not. Consequently t "infectious diseases" will be found to conta obviously general diseases, with some only o pronounced local characteristics. The succe deal with the diseases of the various systems, n cardiac, alimentary, &c., amongst which it will many have an origin in specific infection.

INFECTIOUS DISEASES.

NATURE OF INFECTION.

By infectious diseases is meant the diseases which depend upon the introduction into the body from without of a *virus* or *contagium;* and this contagion or infective agent wherever it can be demonstrated has proved to be a microscopic living being, or *micro-organism*, which can multiply within the body.

That some skin diseases (ringworm, favus) were due to the invasion and growth of minute vegetable organisms has been long known, and these fungi could be seen with no great difficulty by the microscope: they belong mostly to the class of *Moulds* or *Hyphomycetes.* The micro-organisms which are related to the infectious fevers require for their detection special methods of staining, as well as very high powers of the microscope. They belong almost entirely to the class of *Schizomycetæ* or *fission fungi.* These are all minute cellular bodies, devoid of a nucleus, and possess the faculty of being stained, when dead and dried, by certain dyes, such as methylene blue, gentian violet, and fuchsin. They occur in various forms, such as minute spherical or ovoid bodies, called *cocci* or *micrococci;* straight rod-like bodies, called *bacilli;* spiral or screw-like bodies, called *spirilla.* Cocci may adhere together in long threads or chains (*streptococcus*), or in plates (*merismopedia*), or in cubical groups (*sarcina*), or in an irregular manner (*staphylococcus*). *Zooglœa* is a term given to masses of cocci or rods united together by a gelatinous intercellular substance. Some bacteria possess *cilia* or *flagella*, by means of which they impart independent movement to the organism. The flagella are few or many, generally longer than the body of the cells and spirally twisted.

Micro-organisms are reproduced by division; but also, especially in the case of bacilli, they form spores, which are more resistant to destructive agents like heat, draught, and disinfectants than the micro-organism itself. Some of these spores form within the cell itself (*endogenous*); others, especially cocci, have been thought to arise by the conversion of one segment of a chain into a spore (*arthospore* or *joint spore*).

In relation to disease an important distinction has to be drawn with regard to the bacteria found in various diseases, and that is that they are not all the actual causes of the disease. Those which can be proved to actually cause the disease are called *pathogenic bacteria;* others which may be found in association with them, but which flourish in dead and dying animal tissues, and in vegetable and inorganic matter, are called *saprophytic bacteria.* A further distinction is made, since amongst pathogenic bacteria some can only thrive on living tissues, while others can live also on outside the body; the former are called *obligatory pathogenic bacteria*, and the latter *facultative saprophytic bacteria.* And conversely, such saprophytes as cannot thrive in living tissues are called *obligatory saprophytes*, and others *facultative pathogenic bacteria.*

With the first discoveries of pathogenic micro-organisms, it was naturally supposed that the *specific* cause of the disease had been found; but in the sense that each particular organism is peculiar to one disease alone, this has not proved to be true. In some diseases only one pathogenic micro-organism is found, and this may be regarded as *specific*, *e.g.*, in tuberculosis, anthrax, and typhoid fever. In other diseases, such as erysipelas, infective endocarditis, and septicæmia, more than one pathogenic organism have been found. The conditions which an organism must fulfil in order to be regarded as pathogenic were first laid down by Koch; increased knowledge requires their amplification, and they are thus given by Kanthack. A pathogenic specific germ (*a*) must be a parasite or a facultative parasite; (*b*) it must be found invariably in the tissues of an animal dead from or affected with the disease in question; (*c*) it must never under any circumstances occur in other diseases, nor within the normal tissues; (*d*) the organism transmitted from the diseased or dead animal to an affected susceptible animal must reproduce the lesion, and in this second diseased animal the original organism must be found; (*e*) if the organism can be cultivated outside the animal body, then an artificial cultivation inoculated experimentally into a susceptible animal must again produce the disease, and this animal must again contain the organism in its tissues or blood; (*f*) these processes must occur in invariable succession under identical conditions; (*g*) the toxins and poisonous substances obtained from the artificial cultivations must agree chemically and physiologically with those obtained from the diseased animal.

According to Kanthack only anthrax, diphtheria, and tetanus fulfil all these conditions; glanders, tuberculosis, actinomycosis, gonorrhœa, and malignant œdema fulfil all but the last. Diseases in which there is more than one pathogenic organism have been already mentioned. Lastly, no organisms have been separated in the healthy (*isolation*); another is by destroying the virus in the sick person, or in whatever clothes, books, room, or furniture he may contaminate, or in whatever excreta may pass from him (*disinfection*); the third is by so modifying the condition of the possible recipient that he becomes insusceptible to the influence of the virus, even if brought into contact with it (*production of immunity; immunisation*).

Isolation.—The patient should be placed in a separate room, if possible on a separate floor of the house, which may be screened off by a sheet wetted with solution of carbolic acid (1 in 40). Thorough ventilation must be as far as possible kept up, as the dilution of the poison by a constant influx of fresh air is a most important part of the process. All unnecessary furniture, curtains and carpets, clothes, &c., to which contagion may adhere, should be removed from the room. The attendants should be, as far as possible, those who are protected by a previous illness; and it should be remembered that their clothes may convey the disease as they pass from the sick room to other parts of the house, unless such overclothing is changed before coming into contact with others. Only such books, papers, or toys should be allowed in the sick room as may be afterwards burned; and food removed from the sick room should not be partaken of by other people.

Disinfection.—*Disinfection of the Excreta*—In enteric fever or cholera the infective agent is contained in the stools. These should be thoroughly disinfected by thorough mixture with strong carbolic acid solution (1 in 20), mercury perchloride (1 in 500), or sulphate of iron crystals, and left to stand some little time before being thrown away. Destruction by strong mineral acid, with subsequent burial in the earth, has been recommended, but it is not generally possible in large towns. The sputa should be similarly disinfected in enteric fever.

Disinfection of the Clothing.—Linen may be disinfected by prolonged soaking in solution of carbolic acid before washing. Woollen clothes must be exposed to a dry heat of 180° or 200°, and this is best done in special ovens constructed for the purpose, now in possession of most of the local sanitary authorities.

Disinfection of the Patient.—After the patient has recovered, and before he mixes with his friends, he should have several warm baths and be rubbed with carbolic soap. The special treatment required in scarlet fever, on account of the prolonged desquamation of the epidermis, to which the virus adheres, will be described.

Disinfection of the Room.—After the patient has left the room in which he has been ill, it requires to be thoroughly disinfected before it is occupied by others. This is best done with burning sulphur. A pound of roll sulphur, or a sulphur candle, should be kills the micro-organisms, or renders their poisons innocuous—has not been clearly shown. The causes which have been suggested, and which are still under discussion, are: The influence of the febrile temperature (*see* Relapsing Fever); the destruction of the bacilli by leucocytes (*phagocytosis*); the influence of the blood or tissues on the bacilli or their poisons. Of these the last seems the most probable, the view being that the bacilli themselves produce a material which prevents their further growth or development, and thus ends the disease.

Transmission of Infectious Diseases.—This is really a branch of preventive medicine, but a brief notice of it cannot be excluded from a work like this. The infectious diseases having been defined as those in which a virus (or micro-organism) is introduced into the body, it must be here stated that the virus is derived, first, from other human beings ill of the disease, directly or indirectly, as in scarlatina, measles, and many others; or, secondly, from animals, as in rabies, anthrax, foot and mouth disease; or, thirdly, from the soil or other source independent, as far as is known, of the previous participation of other men or animals in the process, as in ague. When transmitted from one human being to another, it may be, apart from experimental inoculation, conveyed in solid tissues, in liquid secretions, normal and pathological, in expired air, and in clothes and other articles. In the majority of cases, as in typhus, smallpox, and diphtheria, the breath is no doubt the means by which the poison is conveyed; in others again, as in scarlet fever, the skin; in others, as cholera or enteric fever, the fæcal discharges; in others, like syphilis, the pus from sores. The exhalations from the breath and skin render the patient *contagious* in the proper sense of the term—that is, that those who are near to the patient for a longer or shorter time run some risk of catching the disease; the fæcal evacuations commonly reproduce the disease by infecting the water or milk which others drink, or, possibly, the air which others breathe; and lastly, pus containing the virus must come [illegible] direct contact either with the mucous membrane or with an [illegible] on the surface of the skin.

As to the period during which the infectious diseases are com-[illegible] there is no doubt that this begins with the appearance of the [illegible] symptoms, and lasts some weeks. It is about two [illegible] and mumps; it may be three or four in measles, [illegible] and typhus; eight or more in pertussis. Scarlet [illegible] and chicken-pox are infectious as long as desqua[illegible] on the body; and enteric fever and [illegible], while the stools continue unhealthy.

[illegible]—There are three ways by which the [illegible] from one person to another, or [illegible]. One is by separating the sick from some diseases is the age of the patient, and this point will be referred to when these diseases are described.

Hereditary disposition may be alluded to here as the converse of innate immunity; it is observed especially in tuberculosis. The appearance of this disease in the offspring of tuberculous parents is mostly due to the transmission of this abnormal susceptibility; in rare cases, possibly, to the actual transmission of the bacillus.

Acquired immunity is that which is imparted in one or more ways to individuals previously susceptible. The most common cause of immunity towards an infectious disease is the fact that the individual has already had the disease. There are but few exceptions to the rule that scarlet fever, small-pox, chicken-pox, measles, and other such illnesses do not occur a second time in the same patient. This protection is probably closely related to the conditions which terminate a given infection in those who are suffering; the altered condition of the blood and tissues which destroys the micro-organisms persists afterwards for many years, or a life-time, and antagonises the influence of any subsequent contagion of the same kind.

A method by which protection may be purposely acquired is the inoculation of the individual with some animal substance possessing special qualities in relation to the disease. This may be weakened or *attenuated* virus of the disease in question—that is, the micro-organisms of the disease; or it may be the same micro-organisms in their full virulence, but in very small amount; or it may be the bacterial products or toxins of the disease without the organisms. The first of these methods has been employed in the cholera of chickens, in anthrax of sheep, and in the human subject in hydrophobia. The means used for attenuating a virus cannot here be given, but that employed by Pasteur to prevent hydrophobia is described in the chapter on that disease.

Vaccination used as a protection against small-pox (p. 71) is also an illustration of the method, since *vaccinia* (or *cow-pox*) is undoubtedly small-pox attenuated by transmission through the cow.

Another method of immunisation results from the principle that if an animal susceptible to a given disease is rendered completely immune (*e.g.*, by repeated injections of culture fluids deprived of the bacteria) the blood-serum of this animal subcutaneously injected into another susceptible animal will render it also immune against both the bacteria and the toxins of the disease. The substance contained in the serum has been called *antitoxin*, and the serum itself *antitoxic serum*.

Still another method of obtaining immunity, at least in animals, is to feed them with the living micro-organisms or with their products.

placed in an earthenware vessel or pipkin, and this should rest on two or three bricks in a large pan of water in the middle of the room. The chinks of the windows should be pasted up with slips of brown paper; the sulphur should be set alight, and the door should be closed and pasted up in the same way as the windows. It is needless to say that no one should remain in the room. After five or six hours the sulphur will have burnt itself out; the room may be entered, and the windows thrown wide open. Subsequently the wall paper should be stripped off and burned, the floor and woodwork thoroughly scrubbed with carbolic soap, or sanitas, and the ceiling whitewashed; or the floor, ceiling, walls, woodwork, and furniture may be thoroughly rubbed with bread, which must be afterwards burnt with all the fragments that drop about.

Immunity.—Persons who are insusceptible to a particular disease are said to be immune, or to have immunity. Such immunity may be partial or complete, temporary or lifelong, innate or acquired. Of the conditions of *innate immunity* little can be said. Some species of animals are immune towards the diseases from which other species suffer; it can be hardly expected that particular races of men, much less classes or individuals, should be immune towards the fevers to which the race as a whole, is susceptible. The nearest approach to an innate immunity is that of the negro towards yellow fever. In a given race, however, susceptibility varies very much. It is a fact observed every day, that of a number of persons exposed to the contagion of a particular disease only a certain number will catch the illness; the rest will escape, even though they are not apparently protected by any of the methods to be mentioned below. Moreover, in those who are affected from the same exposure to contagion, the disease may present very different degrees of severity. It is not only matter of observation, but has been shown by experiment, that susceptibility to infectious diseases is increased by starvation, fatigue, cold, damp, unsuitable diet, and other unfavourable conditions: while a mere local influence [illegible] be in operation when pneumonia or bronchitis is succeeded [illegible] invasion of the lungs. But the working of the law is [illegible] and it is quite certain that the fattest and most [illegible] looking children of a family often suffer from, and succumb [illegible] violent attacks of scarlatina; while others, apparently [illegible] may come off with a mild illness. A special [illegible] diseases is noticed in the case of some [illegible] in women after delivery, and in [illegible] undergone surgical operations; in the [illegible] local wound may be the cause of the [illegible] providing for the contagion a means of [illegible] factor in the susceptibility to a maximum from that time to 7 or 8 in the evening, and then falls to its minimum after midnight.

Registration of Temperature.—The temperature of the body is taken, for ordinary clinical purposes, by means of the clinical mercurial thermometer, which registers the temperature after it is removed from the body, by a portion of the mercurial column being prevented returning into the bulb of the instrument. It is usually about four inches long, so that it can be carried in the pocket. The bulb of the instrument may be placed in the axilla, the groin, the mouth, or the rectum.

In the two former situations it is necessary to see that there is complete contact of the skin with the bulb, and it must remain there sufficiently long for the surface of the skin to attain the temperature of the body generally: from one to three or five minutes suffice, according to the sensitiveness of the thermometer. In the mouth the bulb should be placed under the tongue, and the stem must be grasped by the lips. Under certain circumstances the rectum may be employed for taking the temperature; the bulb is introduced for one and a half inches. The result can be depended on; but it is obviously a method that is not always convenient. It may be well to bear in mind another way of ascertaining the body-heat—that is, by passing urine on to the bulb of the thermometer, but its application is rather limited. As a fact, the axilla and the mouth are most frequently employed. Besides the ordinary clinical thermometer, there is in use a thermometer, shaped like a very small watch, which is sensitive, but has the disadvantage of not fixing its record: and there are surface thermometers, which are useful in special investigations.

In consequence of daily variations, both in health and disease, it is desirable to record the temperatures at least twice a day: the best times would be 5 or 6 A.M. and 5 or 6 P.M., so that the lowest and highest temperatures should be observed. Social arrangements do not usually allow of this in slight cases of illness, so that 10 A.M. and 6 to 9 P.M. are more often the times selected; but it must be remembered that at 10 A.M. the temperature is already rising, and that after 7 P.M. the maximum is generally passed. In severe illnesses, like typhoid fever, pneumonia, &c., it is very desirable to take the temperature at least every four hours, when the daily variations can be more closely watched. The temperature should in all cases be recorded on a *chart*, with a dot for each observation and lines drawn from dot to dot; by this means the oscillations of the temperature are graphically represented, and its general behaviour is more clearly brought to the mind than by long columns of figures. It has been objected that we can never know what takes place between each pair of observations, and that a line drawn, say, from a dot at 99° to another dot at 101° four

In the case of man, these methods of antagonising bacterial diseases have been applied both preventively and curatively.

A true immunity is sought in the case of vaccination and of cholera-inoculations. In hydrophobia the process initiated by Pasteur is employed in the long period of incubation *after* the virus has been inoculated by the dog-bite. The disease is not so much prevented as strangled at its birth. Finally, in diphtheria and tetanus antitoxic serums are used to diminish the effects of disease already declared (*serum-therapeutics*).

The explanation of the above results is still far from complete. It is admitted that immunity does not depend solely on the process of *phagocytosis* or the destruction of the micro-organisms by the leucocytes of the infected person, though this may have an important influence in certain cases; but it is more generally believed that the explanation must lie in the stimulation of the tissues and cells of the body, phagocytes as well as others, to a greater resisting and antitoxic power than they before possessed. In the case of an antitoxic serum this effect is rapidly attained by its injection into the system.

A perfect classification of the infectious diseases is at present almost impossible. It will be sufficient here to make some provisional groupings.

Diseases of more or less certain bacteriology.—Due to Schizomycetes: Relapsing fever, enteric fever, influenza, cerebro-spinal fever, yellow fever, erysipelas, diphtheria, cholera, plague, tubercle, leprosy, pyæmia, septicæmia, tetanus, actinomyces, anthrax, glanders.

Due to Protozoa: Ague, amœbic dysentery.

Diseases of uncertain bacteriology.—Typhus, Mediterranean fever, Weil's disease, scarlet fever, measles, rubella, variola, vaccinia, mumps, whooping-cough, dengue, beri-beri, dysentery, syphilis, hydrophobia, foot and mouth disease.

PYREXIA.

The terms fever and pyrexia are not always used in the same [illegible] being sometimes limited to the mere fact that the [illegible] is elevated, fever comprehending the rise of [illegible] with all the other bodily disturbances [illegible] accompany it. As is well known, the temperature [illegible] between 97·5° and 99° Fahrenheit. [illegible] being the normal temperature, and [illegible] thermometer give such a record. [illegible] of the normal temperature [illegible] between 2 A.M. and 7 A.M., [illegible] 2 in the afternoon, remains at [illegible] becomes deeply congested or livid, and the extremities show the same change.

Alimentary System.—The tongue becomes furred; generally, at first, the fur is white, and the tongue is still moist; then the tongue becomes dry, the fur peels from the edges or tip, and shows the bright red tongue beneath. Later on, the tongue becomes very dry, stiff, hard, dirty brown in colour, fissured on the surface, and caked with dried remains of saliva, buccal secretions, food, and sometimes blood. In this stage also the gums are covered with a similar collection, which is called *sordes.* Loss of appetite, or anorexia, is one of the first signs of fever; sometimes sickness is present, and in all cases digestion is feeble. The bowels are usually constipated. The spleen is often slightly, in certain diseases very much, enlarged and tender.

Circulation.—The heart's action is quickened, at first excited, then feebler. The pulse ranges from 80 to 120 or more. It is at first full, bounding, and firm; it soon becomes softer and dicrotic; and with high fever it is hyperdicrotic (*see* the Pulse). In later stages, as the heart becomes more feeble, it is quick, very small, very compressible, running or flickering. With progressive weakening of the heart's action, the first sound becomes faint, or inaudible, and the impulse may be detected outside the nipple, showing that the heart is becoming dilated.

Respiration.—This is quickened in proportion to the pulse, and the rise of temperature: it may rise to 30 or 40 in the minute. When the illness has lasted some time, the bases of the lungs become congested (hypostatic congestion), and the respiratory movements of the upper part of the chest in front are exaggerated.

Kidneys.—The urine in fever is scanty, high-coloured, and deposits a brick-red sediment of urates on cooling. The urea is in excess, and the chlorides are usually diminished. In severe febrile illnesses, there may be a small quantity of albumen.

Nervous System.—Headache is common at the commencement of pyrexia; there is also a heavy feeling, dulness, or disinclination to think or make any mental effort: after a time, the patient not only does not wish to, but cannot exert the intellect; he becomes drowsy, and when he drops off to sleep, begins to talk. Later on he is delirious without really sleeping, and the delirium may be muttering, and is occasionally maniacal, the patient getting out of bed, struggling with his nurses or attendants, or jumping out of the window. In the last stages there is profound unconsciousness or coma. The disturbance of the muscular system shows itself in general bodily weakness, tremor of tongue or limbs when they are moved, and twitching of the muscles (subsultus tendinum).

Daily Variations.—The temperature in fever shows daily hours later, gives a false impression of a steady rise, of which we have no evidence. But when once we recognise that there may have been variations in the interval, we need not be misled by the chart, any more than by a column of figures recording the same observations, and it is certain that the chart gives at once a comprehensive grasp of the course of the fever so far as it can be known from the observations taken. When studying the four-hour chart of a long illness, I have often found it useful to draw up from it a fresh chart, selecting only the lowest morning and the highest evening temperatures, and thus showing, in the most prominent way, the extreme oscillation in the body-heat on each day.

Range of Temperature.—In disease the temperature ranges from 92·3°, or even lower, to 110° or 111°. Temperatures of 116° and 122° have been recorded, but considerable doubt attaches to their genuineness.

Many terms have been used to denote the different degrees of temperature above or below normal; Wunderlich gives eleven in all. But I think the following are all that are practically wanted:

Collapse temperatures . .	92·3° or lower to 96°.
Subnormal " . .	96° to 97·5°
Normal " . .	97·5° to 99°.
Slight or moderate pyrexia .	from 99° to 101° in the morning, or 102·5° in the evening.
Severe pyrexia	from 101° to 103° in the morning, or 105° in the evening.
Hyperpyrexia	above 105°.

CONDITIONS ASSOCIATED WITH PYREXIA.

Pyrexia, fever, or febrile reaction, is accompanied by many other disturbances besides elevation of temperature; indeed, every function of the body is more or less upset whenever the temperature is raised for more than a very short time; and the symptoms which will be here shortly enumerated, are present more or less in all the febrile diseases.

Skin.—It is hot to the touch, sometimes intensely so; generally dry, but it may be moist; and in some diseases profuse sweats may occur, which, sometimes perceptibly, sometimes scarcely at all, reduce the temperature. Such perspirations may cause an eruption of sudamina, or miliaria. The colour of the skin over the body is generally normal, unless there are eruptions, such as miliaria, or the specific rashes of scarlatina, measles, typhus, and others. But the face is often flushed, especially at the commencement of a fever: often the cheeks and lips are flushed, and the face is elsewhere pale; later on, with a failing circulation, the face as if cold water were running down his back; or he shudders or shivers, and finally he may have a definite *rigor*. This is a more or less prolonged attack of shivering, in which the patient trembles all over, his teeth chatter, he feels intensely cold, his face is pinched, and the nose, ears, and finger tips are livid. But though the surface is cold, the internal parts are hot, and if the thermometer is used, it will be found that the temperature is constantly rising from the first. The rigor may last from a few minutes to half an hour, an hour, or more. In young children, rigors do not occur, but their place is often taken by a convulsion.

The second stage of the fever is the *fastigium*, in which the skin is hot, and the various phenomena already recorded are present.

In some fevers the temperature may rise to its maximum or *acme* just before the onset of the third stage.

The third stage is that of *defervescence* or decline, which occurs either by *crisis* or *lysis*. In *crisis* there is a rapid fall of temperature to the normal within twelve to thirty-six hours; it is sometimes accompanied by profuse sweating, sometimes by diarrhœa (critical sweat or diarrhœa). In *lysis* the temperature falls more slowly, taking three or four days to reach the normal.

For some days after a pyrexia, the temperature may be unusually low (subnormal), *e.g.*, 97° or 96° in the morning, and from this time the period of *convalescence* is established.

The phenomena of rigor, fastigium, and crisis, occur typically in ague, in which they may be all comprised within a period of six to twelve hours.

Death in Fevers.—This results, first, either from exhaustion after prolonged illness or from the intensity of the fever or blood-poisoning in a few days; secondly, from hyperpyrexia in a small number of instances only, though it is not uncommon for the temperature to rise very high when the patient is at the point of death from other causes; thirdly, from cardiac failure; fourthly, from pulmonary complications.

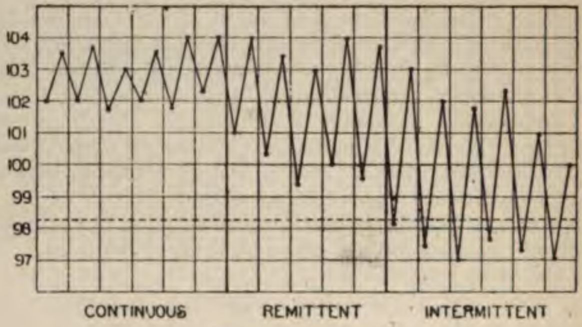
Certain anatomical changes are common to nearly all deaths in high fever. The blood is thin, unduly liquid, dark in colour, and rapidly stains the aorta and large vessels. The red corpuscles are diminished, the white corpuscles are increased in quantity. Small petechiæ or hæmorrhages are found under the serous membranes of the pleura and pericardium. The solid organs—liver, spleen, and kidneys—are large and soft, and the kidneys and liver show, under the microscope, some granular change in their secreting cells.

The muscles may show the peculiar degeneration which was described by Zenker (*see* Enteric Fever, p. 44), and hæmorrhages and abscesses may form in their substance.

fluctuations, which are, as a rule, similar to those observed in health—that is to say, the temperature is lower in the morning, and higher in the evening; the lowest point is commonly reached about midnight or 2 A.M., and the highest from 4 to 6 P.M. Occasionally the reverse obtains, the temperature is highest in the morning, lowest in the evening—*typus inversus*. The pulse and respiration rise and fall with the temperature, and the general discomforts of the patient vary in the same manner.

Varieties of Pyrexia.—The pyrexia which accompanies or constitutes an illness may be one of three kinds: it may be *continuous*, *remittent*, or *intermittent* (Fig. 1). A continuous fever is one in which the temperature is constantly above the normal, and the differences between the morning and evening temperatures never exceed the variation in health—that is, one and a half degree. A remittent fever is also one in which the temperature is constantly above the normal, but the difference exceeds one and a half degree. Thus, in the morning it may fall two or three degrees, but never reaches the normal. An intermittent pyrexia is one in which the morning temperature falls to the normal, or below it, while the evening rise is two, three, or more degrees above it.

FIG. 1.



Types of Pyrexia.

In some cases of illness there may be, at one time, a continuous pyrexia, at another a remittent, and at a third time an intermittent pyrexia; as, for instance, in enteric fever and in phthisis.

Course of Pyrexia.—In many cases of fever a very definite course may be recognised, the beginning and the end being marked by certain characteristic phenomena.

The fever begins with a sensation of cold—it may be a mere chilly feeling, so that the patient seeks the fire, and he may feel

The Cause of Pyrexia.—This is a subject that can only be shortly dealt with here: it has been much discussed, and there is still much uncertainty about it. The normal temperature of the body depends, first, upon *Heat-production* (or *Thermogenesis*) by chemical changes, and it is said that 80 per cent. of heat-production goes on in the muscles, independent of their contraction, and controlled by nerves arising from centres in or near the corpus striatum; secondly, upon *Heat-loss* (or *Thermolysis*), which occurs from the skin to the extent of about 80 per cent., and by the lungs (and the ingesta passed out as fæces) to the extent of 20 per cent. The nerve apparatus concerned is chiefly the vaso-motor system, by which the circulation through the skin is affected. If the skin becomes hot, the vessels dilate, the skin is congested, it becomes a better conductor, more blood passes through it, and so heat escapes by radiation to the surrounding air. With high fever also, respiration is quickened, and the loss of heat from the pulmonary surface is proportionately increased. The centre controlling the operation of heat-loss is situate in the medulla oblongata.

Obviously, the temperature will rise if the heat-production increases with normal heat-loss, or if the heat-loss is less with normal heat-production. Pyrexia was attributed by Traube to the latter alone, by others it has been accounted for by the former alone. Hale White believes that the processes in operation are different in different forms of fever.

It is believed that a regulating function also exists, which maintains the balance between production and loss. It is called *Thermotaxis*, and probably has its seat in the cortex of the brain (Hale White, Ott.). In different degrees of fever, according to MacAlister, all three functions may be disturbed. Heat-regulation, the highest function, is disturbed first, producing slight irregular pyrexia; in a higher degree of pyrexia, heat-production and heat-regulation are both upset, the former being increased; and in hyperpyrexia, heat-loss, the lowest function, is also involved. Burdon-Sanderson regards variations of temperature as depending especially upon heat-loss and upon its regulation, while heat-production is, according to him, always liable to be in excess of actual requirements, so that disturbances of regulation nearly always result in pyrexia, and not in collapse.

The next point is to determine how these functions are disturbed—that is, what are the remoter causes of the pyrexia? Some of these causes are well known. The groups of febrile disorders usually recognised are, (1) infectious diseases, or *specific fevers;* (2) fevers occasioned by local inflammations, called *symptomatic fevers;* and (3) fevers which arise from local disease, or are associated with even functional failure, of the nervous system: the last are often spoken of as *neurotic fevers.*

Stronger Antipyretic Drugs.—These are commonly given in single doses when the temperature reaches a certain height, such as 102° or 103°. The result is that the temperature falls within two or three hours, and, remaining low for a time, rises again in six or seven hours to a height not much different, if at all, from what it would have reached had no antipyretic been given. The following are the drugs which may be so employed and their doses:—Quinine sulphate, 20 to 30 grains; salicine, 30 grains; salicylic acid, 20 grains; antipyrin (phenazone), 15 grains; antifebrin (acetanilide), 2 to 5 grains; phenacetin, 5 to 10 grains. The last three are the most certain in their antipyretic action, but care must be taken not to exceed the maximum doses here stated, as these drugs are apt, especially antifebrin and phenacetin, to cause cyanosis and collapse when taken in too large quantities. Antipyrin occasionally produces an erythematous or urticarial rash, which is of little importance. Antipyrin has been shown to act by increasing the discharge of heat from the surface of the body, doubtless by an influence upon the nervous centres presiding over the inhibitory or dilating vascular nerves.

External Application of Cold.—This may be done in several ways:—The cold bath; the wet pack; sponging; ice applications; Leiter's coils.

Cold Bath.—This has been largely used in the treatment of enteric fever. The temperature is taken every three hours, and whenever it is found at any of these periodical observations to be 102·$\frac{3}{5}$ F. or higher, the patient is placed in a bath of a temperature of 70° F., in which he remains for ten or fifteen minutes, according to its effect upon him. He is then removed, lightly dried, and replaced in bed. The temperature will then be generally found to have fallen to 99°, 98°, or even lower. The system is open to modifications; the observations may be made less frequently, the bath may be only used when the body-heat is 102·5°, or 103°, or 103·5°, and the temperature of the bath may be as low as 60° or as high as 80° or 90°. Sometimes the patient is put in the bath at a temperature of 90°, and ice is then introduced to bring down the heat to 75° or 70°. It is obvious that the greater the number of baths, and the lower their temperature, the greater will be their effect upon the mean body-heat. I have generally made observations every three hours and used a bath of 80°, when the temperature was found at 103°.

Brand

temp. taken [illegible] Bath [illegible] 20 minute.

[illegible] 8 [illegible] 24 [illegible]

Wet Pack.—A sheet is wrung out of ice-cold water and wrapped round the patient for ten or fifteen minutes, the application being made under the same conditions of bodily temperature as are directed for the bath.

Sponging.—The body is uncovered and sponged over with cold or ice-cold water from seven to ten or fifteen minutes. This method is not generally so effective as the two former; the temof the insane. (6) Irritation of sensory nerves, as in intestinal strangulation, renal and hepatic colic, perforations of the intestine, and surgical operations. (7) Extensive skin affections, such as universal eczema, and large burns. (8) After fevers, when the temperature may long remain subnormal, or in the course of pyæmia. (9) Poisoning by phosphorus, atropine, morphine, carbolic acid, and alcohol; uræmia and diabetic coma.

Treatment of Pyrexia.—The special treatment of each particular case will depend more or less upon the cause; but the general principles of treatment are as follows:—The patient should be at rest in bed, in a well-ventilated apartment; he should be kept cool, the amount of clothes being lessened if the fever is very high. A distinct lowering of temperature may be sometimes effected in this way, a point to be remembered all the more as the tendency of the patient's friends is to heap clothes upon him to prevent his "catching cold." The extremities, however, must be carefully watched, and specially covered or warmed, if necessary. The diet should be light, digestible, and nutritious. In the majority of cases milk is the most suitable, and an adult patient may take from two to four pints in the twenty-four hours; beef-tea, mutton-broth, chicken or veal broth, may generally be substituted to the extent of half or one pint in twenty-four hours. In cases where milk disagrees, or is felt to load the stomach, or is rejected, it may be mixed with half its bulk, or an equal quantity, of barley-water or soda-water; or it may be peptonised or predigested by warming for a little time with liquor pancreaticus. These foods should be given in small quantities at short intervals; for instance, five ounces of milk every two hours in severe cases. In many cases of pyrexia the temperature may be directly dealt with by methods known as *antipyretic*. It must be distinctly understood that such treatment will not shorten the duration of the illness; that in many illnesses the temperature will of itself fall in a few hours, that is, in the early morning; and that there is very rarely (except in rheumatic hyperpyrexia) any danger that the temperature will rise to a height which can be directly fatal. But efficient antipyretic treatment certainly increases the comfort of the patient during the time that each successive dose or application is in operation, and possibly, in some diseases, diminishes the risk of damage to viscera; and although such a treatment does to a certain extent interfere with the natural course of the temperature, it need not materially falsify our estimate of the progress of the illness.

Antipyretic methods may be divided into three groups:—

Milder Refrigerants.—These are the ordinary saline remedies—citrate of potassium, acetate of ammonium, dilute acids, which were formerly given in every fever, but have very little influence.

by clothes and other similar articles, but does not seem to be exhaled from the dead body, nor carried by water, milk, or food.

It attacks persons of all ages, and males and females equally. Those who have already gone through an attack are, with rare exceptions, protected from another. It is not much affected by season or weather, except in so far as they may determine overcrowding; but it is confined to temperate and cold climates. It is at present comparatively rare in England.

Symptoms and Course.—The period of *incubation* is variable; in a few instances it has been two days or less; but in more cases it is twelve, thirteen, or fourteen days. The disease begins, like many other fevers, with headache, loss of appetite, and a general feeling of illness, with perhaps some chilliness or actual rigor. In severe cases there are sharp rigors, with nausea or sickness. During the next two or three days, while yet there is nothing distinctive of the disease, all the symptoms of severe fever are rapidly developed. The temperature rises to 103°, 104°, or 105°, the pulse and breathing are proportionately quickened; there are furred tongue, continued headache, flushed face, and suffused eyes; pains in the back and limbs, anorexia, scanty high-coloured urine, and constipation. By the third or fourth day the patient is generally so ill as to be obliged to take to his bed. On the fourth or fifth day, sometimes as early as the third, sometimes on the sixth or even the seventh, appears the characteristic eruption, or *mulberry* rash, of typhus. It comes out on the abdomen and chest, and on the backs of the hands and wrists, and in the course of two or three days covers the trunk, and perhaps also the arms and legs. The face and neck are mostly free. It consists of two portions: one, a dusky red mottling, fading on pressure, not giving rise to any elevation of the surface, and often described as "subcuticular"; the other, a rash, consisting of bright or dark-red papules, slightly elevated above the surface at first, and having some resemblance to the rash of measles. These "maculæ" at first fade on pressure, but after a day or two they become more dusky, and later some of them become petechial from extravasation of blood, which persists under the pressure of the finger.

The rash gradually fades during the second week, by the end of which it is generally gone. In rare cases the mulberry rash is preceded by a diffused red rash, or *roseola*, not unlike the eruption of scarlatina; but this disappears entirely before the mulberry rash comes out. By the time the eruption is developed —that is, at the end of the first week—the fever has made progress. The patient lies on his back in bed, with a dull, heavy, stupid look, the face flushed, conjunctivæ injected, and pupils contracted. The temperature, pulse, and respiration are, of course, above the normal; the tongue continues furred. He perature commonly falls from one and a half to two degrees only.

Ice-bags.—These may be placed on the chest or abdomen for varying periods.

Leiter's Coils.—These are closely wound coils of metal tubing, through which a continuous stream of cold water is passed. They may be applied to the head, chest, or abdomen.

Stimulants.—In all severe febrile illnesses the necessity for temporary stimulation may arise; this may be supplied by sal volatile, carbonate of ammonium, or small doses of ether; but more certainly by brandy or other distilled spirit. Signs of nerve-prostration or cardiac failure are the indications for the use of stimulants—quick, feeble pulse, inaudible first sound of the heart, dry tremulous tongue, or delirium. A pulse under 100 rarely requires stimulants. If the heart becomes irregular or shows evidence of dilatation, digitalis and ammonia or strychnine should also be given. The quantity of brandy may be from two to six or eight ounces in the twenty-four hours; but the larger quantities must not be continued for many days, and especially in prolonged illnesses like enteric fever, one should carefully watch the effects of this drug, since an excessive amount will keep up a quick pulse, and a drowsy, muttering delirium, deceptively like the very condition for which it was originally given.

- The reason these contagious diseases attack children so, is because they are not protected by former attacks

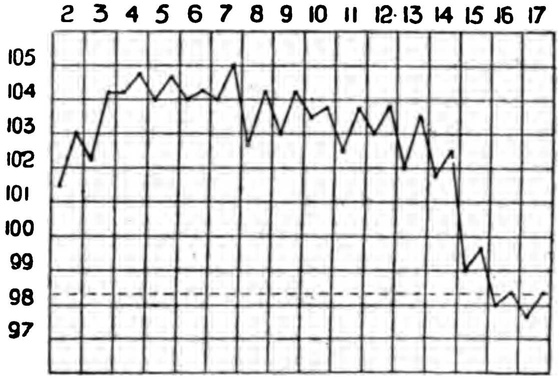
TYPHUS FEVER.

(*Spotted Fever, Putrid Fever, Jail Fever.*)

Typhus fever is an acute specific contagious disease, lasting two or three weeks, and characterised by a typical eruption.

Ætiology.—Typhus occurs, for the most part, in epidemics, which may last for some months, and then gradually subside. These epidemics commonly break out in large towns, attacking especially the poorer quarters, where overcrowding, filth, and insufficient food appear to act as predisposing causes of its spread, rendering those subject to such conditions extremely susceptible to the influence of the poison, as well as especially fit to further its development. It has also, at different times, raged in prisons and in armies in the field. But, even to those not so prepared, typhus is very contagious, and medical men and nurses attendant upon typhus patients, and much in contact with them, are frequently attacked; and it is especially during the second week, when an offensive odour is given off from the patient, that the disease is thought to be most contagious. The poison is, however, rendered innocuous by free dilution with air. It is conveyed occurs the rapid fall which constitutes the *crisis* of the disease. This happens in the large majority of cases about the end of the second week—that is, from the thirteenth to the sixteenth day (Fig. 2), or it may be earlier. The temperature then falls four or six degrees in the course of twenty-four to thirty-six hours, from 103° it may be, or higher, to 99°, 98°, or lower still. At the same time, the pulse and respiration become slower, and the crisis is not uncommonly accompanied by other indications of change in the condition of the patient—viz., profuse sweating, an attack of diarrhœa, or the appearance of an abundant deposit of urates in the urine. From this time the recovery of the patient is remarkably rapid: he soon regains consciousness, the tongue cleans, the dusky hue of the face subsides, and thirst is replaced by appetite. The temperature commonly remains normal during convalescence, but the pulse is often rapid, 90 to 100, for some days after the crisis. Though at first excessively weak, and continuing to emaciate even after the crisis for a day or two, the patient quickly gains strength and flesh, and may be, in the course of a month or six weeks, better and stouter than he was before the fever.

Fig. 2.



Temperature in a Case of Typhus Fever.

But the termination is often unfavourable; in fatal cases, death takes place commonly towards the end of the second week from cardiac failure, or from congestion of the lungs, or pneumonia, or from increasing coma. It is occasionally preceded by a rapid rise of temperature.

Morbid Anatomy.—The *post-mortem* appearances are scarcely distinctive, but are such as are characteristic of severe fever. The

are dark red or
able, yielding blood and serum
granular surface of pneumonic he
is, however, also present in some ca
soft and somewhat enlarged, and tl
large, though sometimes quite norm
results of old disease. The enlargen
is associated with cloudy swelling an
tion. No definite micro-organism ha
the cause of typhus fever.

Among the **Complications and Seq**
often arises during the second week, a
of convalescence, delaying recovery, a
gangrene; bed-sores; gangrene of tl
pudenda, probably from embolism; su
inflammation and suppuration of the
sublingual glands; and erysipelas of the
femoral vein, with resulting œdema of t
less frequent than in enteric fever. Me
a few instances *post-mortem*, and uræm
occur, in association with renal disease a
either of old date, or have been set up l
Mania sometimes occurs during convales

Pneumonia
B. sores
Gangrene
[illegible] suppuration
[illegible]
Erysipelas
[illegible]
[illegible]
[illegible]
mania

Diagnosis.—At the outset of the dis
to distinguish it from other febrile illn
that the patient has been exposed to con
tion appears it may be mistaken for m
measles generally comes out first on th
spots are brighter red, more raised, mo
perhaps arranged in crescentic forms,
preceded by catarrh. The eruption sho
guish it from acute meningitis and f
which typhus may be mis

measles

n the profuse sweating and the sudden cessation of fever.
e cases a second relapse occurs, and in some even a third,
or fifth. But these last form a very small proportion of
s. On the other hand, some patients escape without any
t all. Most commonly the relapse is of somewhat shorter
than the first attack, lasting on an average four to five
t it may be, only two or three days. It is often milder
first attack, but it may be more severe; and, indeed, a
proportion of deaths take place during the relapse.
es it is indicated only by slight rise of pulse and tem-
, and general malaise. Convalescence from relapsing
often slow. The disease is much less fatal than either
r enteric fever, the mortality, according to Murchison,
per cent.; death occurs most commonly at the height of
attack, or immediately after the crisis, from exhaustion
apse; and this is especially the case with old people.
me epidemics suppression of urine and convulsions have
death; and a fatal result may follow such complications
he as pneumonia, dysentery, or parotitis. From the
ation of each febrile attack bed-sores are not frequent or

ications and Sequelæ.—An important complication is
ia, which has been frequent in some epidemics, and may
ause of death; it may be associated with pleurisy, and
ngrene of the lung has resulted. The spleen may attain
size, and it has been known to rupture, with a fatal
Diarrhœa and dysentery sometimes occur in a severe
aundice has been already referred to; it occurs in
less than 20 per cent. of the cases, and may appear in
attack alone, in the relapse alone, or in both paroxysms.
the cases in which it occurs are severe, or even fatal, but
e quite mild. In the severer forms it may be accom-
epigastric and hypochondriac pain, vomiting of blood,
ria, hæmorrhages, delirium, coma, and subsultus.

pulse, serious pulmonary complication, or much low delirium. Maniacal delirium, however, seems to be aggravated by their use. From four to six ounces of brandy may be given in twenty-four hours, in divided doses; but very severe cases may require as much as ten or twelve ounces. Particular symptoms may require to be treated. If there is constipation, it can be relieved by a dose of castor oil. The bladder should be constantly watched: while he is still conscious the patient may be made to pass his urine, but in later stages the catheter may become necessary. If there is much headache, sleeplessness, or delirium, cold may be applied to the head by means of an ice-bag or Leiter's coils; but opium is often required to procure sleep. It may be given as tincture or the liquor opii sedativus, or morphia may be injected subcutaneously; thus a grain of opium or a quarter of a grain of morphia may be given at night in the first week. Chloral may also be given under similar circumstances, and is more suitable when the delirium is maniacal. In the later stages of the disease, sedatives are less desirable, and coma, severe lung complications, and suppression of urine decidedly contra-indicate their use. For lung complications, carbonate of ammonium is the best remedy; turpentine is also of value, and mustard or linseed poultices may be applied to the back and sides of the chest. When the temperature is very high, sponging the body with tepid water will often give temporary relief to the sensations of discomfort which the patient suffers; but the systematic employment of cold baths to reduce the temperature, such as have been used in enteric fever, has not been found to give very satisfactory results. On the third day of convalescence, if the tongue be clean, solid food may be given; and the stimulants required in the height of the fever should be gradually diminished.

RELAPSING FEVER.

(*Famine Fever, Febris recurrens.*)

Relapsing fever is a specific contagious disease, generally occurring in epidemics, not distinguished by any rash, but consisting of a short fever which terminates suddenly in six or seven days, and is followed by a relapse of the same nature after an interval of about a week.

For some time this disease was confounded with typhus fever. It occurs under conditions almost precisely the same—namely, overcrowding, destitution, poverty, and starvation, and has often actually co-existed with typhus. But Dr. Henderson, in 1842, and Sir W. Jenner, 1849–51, pointed out the differences between them, and the two diseases have since been regarded as distinct. This view is confirmed by the discovery of a micro-organism in the blood of patients with relapsing fever, which has never been found in typhus.

Ætiology.—Relapsing fever attacks patients of all ages, and of both sexes, though male patients have been more numerous, in the proportion of three to two. The occurrence of epidemics is very little affected by season or weather, but has an obvious connection with overcrowding and destitution, and with the occupation of the patient, in so far as it is one which the poorer classes of the population commonly follow. Thus hawkers, beggars, and tramps are frequent amongst those affected by this disease; and it has often been shown that such patients were at the time in a state of great destitution, or half-starved, being driven to eat raw vegetables, turnips, rotten apples, or unripe fruit. Indeed, its connection with starvation is so obvious that the name "Famine Fever" has been given to it. On the other hand, of people in a better position, those only are affected who are brought into direct contact with the disease in hospital or elsewhere, such as doctors, nurses, and clergymen. Thus the disease is distinctly contagious, even amongst healthy people, but the conditions of destitution and starvation are powerful predisposing causes. The poison may be conveyed through the air, or communicated by means of clothes, bedding, &c. It has also been conveyed both to the human subject and to monkeys by inoculation of the blood of a patient suffering from the fever. Like typhus, it is rendered innocuous by free dilution with pure air.

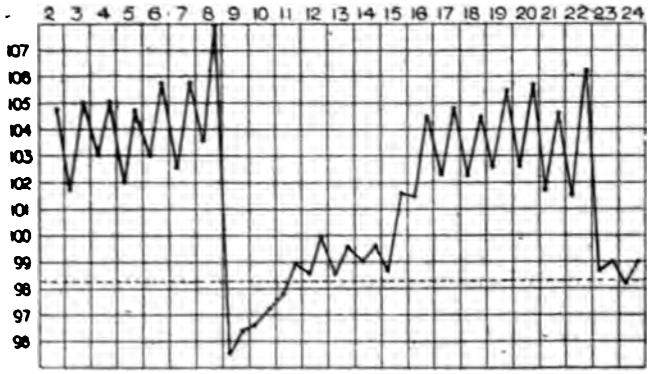
Symptoms and Course.—The period of *incubation* varies from one to sixteen days, but in a large proportion of the cases examined for this purpose, it was less than nine days. At this time there are generally no symptoms. The fever begins suddenly with chill or rigor, and this is accompanied by frontal headache, and pains in the back and limbs. After a short time the chill is succeeded by a feeling of heat, the skin becomes dry and burning, and the headache and pains in the limbs are aggravated. If the temperature be taken on the first day it will be found to be 102·5° or 103°, or higher. The patient soon takes to his bed, and suffers severe thirst, with anorexia, and perhaps nausea and vomiting, the face is flushed, and the tongue is covered with a thick white fur. The temperature continues high, reaching 104°, 105°, or 106°, at night, though often falling a degree in the morning.

There may be with this an occasional rigor, and not infrequently free sweating. The pulse is rapid, 110 to 120, and the respirations are quickened to 30 or more. In some cases there is jaundice, which may be very pronounced: in such cases the urine is stained with bile, but the fæces are normal in colour. There is frequently tenderness over the hepatic and splenic regions, and both liver and spleen are enlarged, the latter more often and more decidedly than the former. Occasionally there is well-marked herpes of the lips.

The urine is very variable, its quantity being affected by food, vomiting, or sweating. In the height of the fever, the chlorides are diminished, and there is occasionally a small quantity of albumen. Epistaxis sometimes occurs. And though there is no typical rash, in rare cases an eruption has been observed, either of pink maculæ or of petechiæ.

The condition remains much the same for about a week; the patient, as a rule, gets but little sleep, suffers severely from the muscular and arthritic pains, and is generally quite clear mentally, till towards the end, when he becomes delirious. All the febrile conditions then become greatly aggravated, the pulse quickens to 130, the respirations to 40, the temperature rises in a few hours to 106°, 107°, or even 108°, the face is flushed, the tongue dry, or brown, and the delirium is increased; and then suddenly the crisis takes place. Perspiration breaks out, rapidly becomes profuse, and the temperature, pulse, and respiration quickly fall. In a few hours the temperature is 98°, 97°, or 96° (Fig. 3), the pulse 70 or less, the skin is moist, the tongue clean, and the patient free from delirium; and except that he feels weak, may express himself as being comparatively well. However, where the fall of temperature is considerable—and it may be 10° or 12° in twelve hours—and the sweating is profuse, there may be considerable collapse for some hours, especially in elderly patients. The crisis is sometimes marked by diarrhœa or by epistaxis. Recovery from this point is very rapid; the temperature, which has become subnormal, regains the normal level; the patient is soon raven-

FIG. 3.



Temperature in Relapsing Fever.

defects in regard to this disease, lies only in the probability that the individual who constantly breathes sewer gas will have the bodily resistance so lowered that he will readily succumb to the disease if exposed by other means more certainly proved to be effective. Drinking water is, however, not the only source of danger. In 1870 an epidemic of enteric fever occurred, in which nearly all the patients affected were found to have been supplied with milk from one particular dairy. There was every probability that a tank in the cowyard was contaminated with sewage, and that water from the tank, used for washing the milk vessels, had infected the milk. Other instances of a similar kind have occurred since. It is also on record that enteric fever has been communicated by diseased meat; and it appears almost certain that oysters supplied from beds exposed to contamination by sewage material, and eaten raw, may convey enteric fever to their consumers.

An important question is, as to the period, after evacuation, at which the stools become infectious. It appears clear that they are not dangerous when fresh; but that it is during their decomposition, after a certain interval, that infective material forms in them; and, although this is generally believed to require three days, it appears possible, from cases observed by Dr. Cayley, that the interval may be as short as twelve hours. Thus, the mere inspection of stools, or their removal from bed to closet, is not dangerous; but bedding soiled with the fæces, and left for some time unchanged, may be the cause of the fever in a nurse, or laundresses may catch it from soiled linen sent them to wash. Probably in no other way do bed-linen and clothes serve to convey the infection.

Symptoms and Course.—The period of *incubation* of enteric fever is variable; but many cases in which it has been ascertained have shown it to be about a fortnight, or between ten and fifteen days. In exceptional cases it may be as short as five or as long as twenty-two days. The beginning of the disease is often very little marked. The patient feels ill, depressed, unfit for work; he has headache, pains in the limbs and back, loss of appetite, and perhaps nausea. These may come upon him so that he scarcely knows when they began; but he can frequently fix a day on which he says he first felt ill. Often the headache is severe, and forms the most prominent complaint. There may be diarrhœa in the first few days; sometimes on the first feeling of illness a purgative is taken, and the bowels continue loose. The patient may go about, struggling to do his work, for five or six days, but generally towards the end of the week he is obliged to give up, and take to his bed. The temperature has been stated, in the first four or five days of enteric fever, to rise two degrees each evening, and to fall one degree each morning, so that at the end and external evidence of the spread of the disease (inflammatory stage). Recovery is tedious. It has been observed that pregnant women almost invariably abort, and in such cases hæmorrhage from the uterus may become a danger.

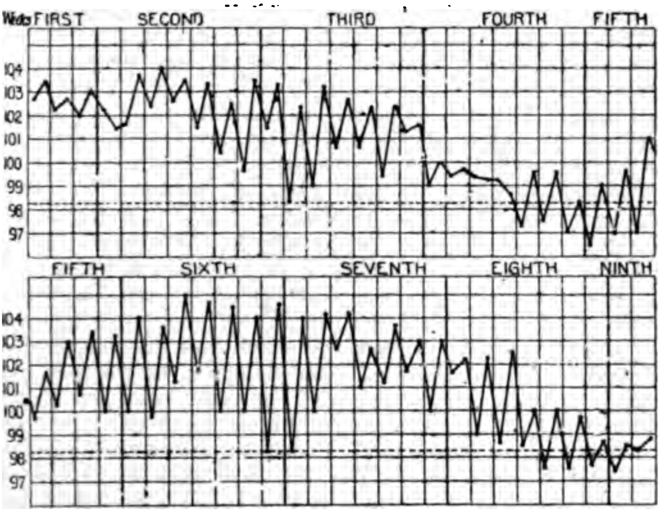
Morbid Anatomy.—With the exception of the condition of the blood, presently to be described, there is no specific or constant lesion. The spleen is, however, very often enlarged, especially when death takes place during a paroxysm: sometimes it presents infarctions; and, rarely, small abscesses have formed in it. The liver also is usually found enlarged, firm, and loaded with blood, but the jaundice is not always explained by any alteration of its structure, or by obvious obstruction of its duct. The kidneys may be congested. Other pathological conditions have been alluded to in the description of the complications.

The micro-organism of relapsing fever is a spirillum, which was first discovered in the blood by Obermeier in 1873. It has been called *spirochæte Obermeieri*. Vandyke Carter found it constantly in those suffering from relapsing fever in Bombay, and proposed to rename the disease *spirillum fever*. The spirillum consists of an exceedingly fine thread, from 16-40 μ in length ($\frac{1}{1500}$ to $\frac{1}{600}$ inch); it is spirally coiled, and in constant movement of a rotatory or lashing character. Spirilla often adhere to one another so as to form masses, or they may adhere, either singly or many, in a tufted manner to the blood-corpuscles. Although there does not seem to be a constant relation between their number and the severity of the attack, their quantity in the blood varies with the different stages of the illness. They are always present during the paroxysms, first appearing, according to some, about the second day; according to others, in the stage of incubation for forty-eight hours before the onset of fever. They frequently increase in numbers as the fever progresses, but about the time of the highest temperature, just preceding the crisis, begin to diminish; by the termination of the crisis they have completely disappeared from the blood, and they remain absent until near the advent of the relapse. In monkeys affected with this disease, Metschnikoff discovered that when the spirilla disappear from the blood, they can be found in the spleen, for the most part included within, and perhaps being destroyed by, the leucocytes (*phagocytosis*). But some of the cells are undergoing degeneration, and it is suggested that spirilla thus escaping may again multiply and cause the relapse. The occurrence of spores has not been shown. The blood also often contains large protoplasmic masses, and free granules.

Diagnosis.—It may be confounded with typhus or enteric fever at first, but the differences become pronounced within a few days. In the course of an epidemic, the sudden severe onset, absence of eruption, severe pains in the limbs, and jaundice, when present, but age has a marked influence, and the disease is much more frequent among young people. Thus, in a number of cases collected by Dr Murchison, one-fourth of the patients were between fifteen and twenty years of age, two-thirds were between ten and twenty-five, and three-fourths of the whole number were under twenty-five. The disease does nevertheless occur in quite old people as well as in the very young. It is more prevalent in the latter part of the year—that is, in the four months August to November inclusive—and cases are more numerous during hot and dry weather than under the opposite condition. Though contagious in ways that will be presently discussed, it is not affected by overcrowding, poverty, and destitution, nor by the emanation of the patient, in the same way as typhus and relapsing fever. It appears to be well established that nothing is given off from the body or the breath of the patient to the surrounding air which can convey the disease to another individual: so that doctors, nurses, and students in hospitals do not take enteric fever directly from the patients. On the other hand, one case may be the means of conveying the disease to a whole town or district, and it has been conclusively shown, in a large number of instances, that such an occurrence is brought about by means of the stools of the patients, which are allowed to contaminate the air or the water supply of those who subsequently fall ill. There is nearly always sufficient opportunity for this in the imperfect means usually employed for the disposal of sewage. In country districts wells used for drinking water may be poisoned in consequence of the soil being saturated with sewage which has leaked from a neighbouring privy or imperfectly constructed cesspool. In one case a well was contaminated by the slops from a laundress's house leaking into it; enteric fever broke out in the houses supplied by the well shortly after the laundress had received some linen soiled by the discharges from a patient with this disease. Where the drinking water is conveyed by pipes, the disease may find an entrance, if the pipes by any chance are defective, and if they lie in a porous soil sufficiently close to any collection of sewage, imperfectly confined, which has received any enteric stools; and a whole reservoir may be infected in the same way. In large towns provided with the water-carriage system common in England, the underground system of sewer pipes exposes many houses to the risk of impregnation with sewer gas, which may either escape from defective soil-pipes into the basement of the house, or into particular rooms, or may force the water-closet trap when the soil-pipe is unventilated, or may reach the drinking-water cistern, the overflow pipe of which may wrongly communicate with the sewer or soil pipe. But it is generally believed now that enteric fever is rarely, if ever, conveyed directly in this way; and that the danger of these sanitary lengthened periods of coma and other severe symptoms, the temperature slowly returning to the normal, and convalescence being very protracted.

Relapses.—A true relapse of enteric fever occurs in a certain proportion of cases, which have been found by different observers to be from 3 to 10 or 11 per cent. It consists of a repetition of all the phenomena of the illness, ulceration of Peyer's patches, fever, diarrhœa, and rose spots; and it occurs after an interval which may be as long as eleven days from the termination of the original fever, but is often much less. Sometimes, indeed, there is no interval of actual apyrexia, and the relapse seems to be continuous with the primary fever. Its duration is often quite as long as that of the first attack (Fig. 5), and, as a rule, it is somewhat milder. Death may, indeed, take place in the relapse, but this is more often from complications, such as perforation of the intestine and peritonitis, or from hæmorrhage, than from the severity of the pyrexia alone. Occasionally a second relapse occurs after another interval of apyrexia; and even third and fourth relapses have been observed, though very rarely.

Fig. 5.



Temperature in a Case of Enteric Fever with Relapse.

Morbid Anatomy. — The essential lesions of enteric fever occur in the *Peyer's patches* and *solitary glands* of the small intestine. These become infiltrated with lymph-corpuscles, and of that period it will have reached 103° or 104°. So many cases escape accurate observation in the early days that it is not always easy to confirm this, but it is certain that in some cases the thermometer may rise on the first evening of illness to 103° or higher. The high level of 103° to 104° once reached, the temperature commonly remains at nearly the same level till the tenth to the fourteenth day—oscillating, however, between morning temperatures of 102° to 103°, and evening temperatures of 103° to 104·5°. Pulse The pulse is quickened, and is full, soft, and markedly dicrotic. Though in some cases very rapid, it is generally, in relation to the temperature, much slower than in typhus and many other febrile conditions: it may never exceed 100, and a pulse of 80 may co-exist with a temperature of 102° or 103°. The respirations are increased in frequency, and there is very frequently slight bronchitis, indicated by sibilant rhonchi, and accompanied, it may be, by mucous expectoration. About the seventh to the tenth day, the patient commonly begins to present the characteristic appearance of enteric fever. He is dull, listless, apathetic, but not so dull and stupid as in typhus; the eyes brighter, the pupils often dilated; the face pale, with flushed cheeks and dark lips; the tongue dry, with a band of dry white fur on each side, the sides, tip, and middle clean and red. Pain in the head may still continue a prominent symptom, and lead him even to cry out. Occasionally profuse perspiration occurs, or bleeding from the nose. At the end of the first week, or later—that is, from the sixth to the twelfth day—appears the characteristic ***rose-rash*** of enteric fever; it consists of rose-pink spots, circular, slightly raised above the surface, flat, convex, but not pointed, so that they are often described as *lenticular*, from two to four millimetres in diameter, disappearing under firm pressure with the fingers, and never petechial like the typhus rash. They are seen first on the abdomen and front of the chest, and may be confined to these parts; but also occur on the sides, back, and the upper arms and thighs. In number they vary from half-a-dozen to twenty or thirty, but they may be much more numerous, and in a certain number of cases (10–20 per cent.) are entirely absent. Each spot has a limited duration, gradually fading in three or four days; but successive crops of spots come out day after day until the end of the third week, or in some cases even later. They are not visible after death. In the second week also the ***intestinal symptoms*** become prominent. The abdomen is generally full, even distended, and resonant on percussion; and there may be both tenderness and pain, but the former is more common than the latter. Pressure in the right iliac fossa, over the seat of the cæcum and lower end of the ileum, often elicits a little pain, and sometimes a sensation to the hand of *gurgling* or of dull crackling, like what is felt when air escapes in the subcutaneous tissue after change is most common in the adductors of the thighs, and in the recti abdominis; and it is sometimes accompanied with hæmorrhage, or the broken-down muscle may form a kind of abscess. The ***lungs*** are either œdematous, or congested at the bases; or, in occasional cases, there is actual pneumonia.

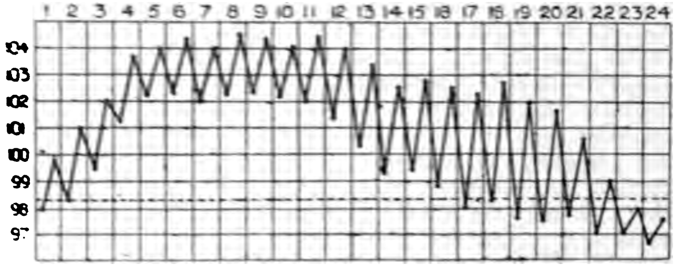
The specific micro-organism of enteric fever is a bacillus discovered by Eberth. It is 2–3 μ in length, with rounded ends, and provided with from eight to twelve fine flagellæ of about twice its length. It bears a close resemblance to the bacterium coli commune, but can be distinguished from it by bacteriological tests. Eberth's bacillus has been found during life in the stools, in blood drawn from the spleen, in the urine, in the sputum, and in the pus of abscesses resulting from periostitis and some other similar lesions. After death they have been found in Peyer's patches, in the mesenteric glands, spleen, liver, kidneys, and meninges.

Complications.—The most important complications are those connected with the intestinal lesions. *Peritonitis* is a frequent cause of death. It arises most commonly from perforation of the floor of one of the ulcerated Peyer's patches, through which the contents of the bowel are extravasated into the peritoneal cavity; but it occasionally happens from extension of inflammation through the peritoneal coat, without any perforation being discovered; and it has also been caused in rare cases by the softening of inflamed mesenteric glands and infarctions in the spleen, and by rupture of the gall-bladder. Perforation of the bowels takes place in about 10 per cent. of fatal cases of enteric fever. It commonly occurs during the third, fourth, or fifth week. Its onset is often accompanied by acute pain, collapse, and perhaps rigors; the abdomen becomes distended and tender, the pulse is small and rapid, and a fatal result soon ensues. But its advent may only be marked by collapse and increased distension; and in very severe cases, with much distension of the bowel, as well as coma and delirium, there may be no certain sign to indicate peritonitis, so that perforation and peritonitis are occasionally found *post mortem* when not suspected during life. It is important to remember that so long as the ulcer remains unhealed there is a possibility of a rupture taking place; and that such a rupture may be induced by any disturbance of the bowel, as by vomiting, defæcation, the exertion of sitting up, or the administration internally of indigestible food or of aperients; and thus, even cases which are running a mild course may have a fatal termination from this cause.

A slight amount of *bronchitis* is frequent in enteric fever, but occasionally it is so severe as to constitute a very serious complication. The face may be quite livid, and a more or less venous tinge may be given to the whole surface; the chest is filled with râles and rhonchi, and there is expectoration of he may acquire an appetite before the fever has entirely left him.

On the other hand, the graver cases are mostly accompanied by a marked increase in the intensity of the nervous symptoms, to which the symptoms of cardiac failure, or severe abdominal troubles, may be added: more or less continuous delirium may supervene, with drowsiness, or even coma, extreme muscular prostration, subsultus tendinum, and plucking at the bed-clothes. The face becomes dusky, the tongue dry, sordes collect on the lips and teeth, the pulse is rapid, soft and dicrotic, the heart sounds are feeble, and the bases of the lungs are congested, as shown by râles and a very feeble respiratory murmur. The urine may be retained, or both fæces and urine are passed unconsciously. The condition resembles that described under typhus fever: the patient is indeed in a truly *typhoid* state. The delirium is less often violent than in typhus, but occasionally patients get out of bed, or refuse food. Cardiac failure shows itself by the feebleness of the heart's beat, the indistinctness of the sounds, the small, rapid pulse, and by venous congestion of the face and extremities, and of the bases of the lungs. Sometimes there is evidence of cardiac dilatation in displacement of the impulse outwards, and the pulse may be irregular or intermittent. With the increase of the nervous symptoms, the abdominal troubles are often prominent, the diarrhœa becomes profuse, and the abdomen is much distended, tense and tender; in this stage the ulcerated bowel may give way, and peritonitis may result from the escape of fæcal matter into the cavity of the abdomen. In a few cases, severe bronchitis is the main feature of the disease: the face is livid, râles and rhonchi are heard over the whole chest, and breathing is seriously obstructed. Under these various circumstances death may occur almost at any time after the tenth or twelfth day; but recovery occurs after

FIG. 4.



Temperature in Enteric Fever.

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a Peyer's patch so affected swells, and projects one or two lines upon the inner surface of the intestine; it is grey, fawn-coloured, or pink, but the surrounding mucous membrane may have its natural colour. The lymph-corpuscles at first multiply in the follicles, but subsequently infiltrate the mucous membrane above and the deeper structures below. As the patches become larger they acquire a creamy-white colour, and about the tenth day or a little later they begin to ulcerate, or slough, presenting at first a superficial abrasion at one point of the surface, which becomes deeper and deeper until a great part of the gland is removed; or a whole patch may slough at once. When the slough is still adherent, it often has a yellow colour from absorption of bile. By these processes the muscular coat or the peritoneal covering may be exposed in the floor of the ulcer, and finally, the peritoneum may slough, ulcerate, or tear, so that the contents of the bowel escape into the peritoneal cavity, and set up a fatal peritonitis. The state of ulceration generally occupies part of the third week, and towards the end of that time, in favourable cases, the process of healing by cicatrisation begins. Ulceration does not necessarily occur. In mild cases the inflammatory swelling subsides without further destructive change. The number of Peyer's patches affected is very variable, and though the cases with severe diarrhœa generally have extensive inflammation of the bowel, there is no necessary correspondence between the extent of ulceration and the severity of the other symptoms. The patches near the ileo-cæcal valve are those first attacked, and the process spreads upwards. The change in the solitary glands of the lower end of the ileum is of the same kind, and in some cases the solitary glands of the large intestine (mostly the cæcum) are also enlarged and ulcerated. Coincidently with these lymphatic structures of the intestines, the *mesenteric glands* are inflamed; they are enlarged, fleshy, pink, red, or purplish, and their histological changes resemble those of the Peyer's patches. Sometimes they break down so as to contain one or more small collections of fluid resembling pus, which in rare cases burst into the peritoneum; but more generally they become cheesy or calcareous. The *spleen* is commonly enlarged, dark in colour, and in later stages of the disease, softened. The *liver* is often hyperæmic, and softer than natural; the *kidneys* are congested, and in both these organs the gland cells are granular. The *heart* is often soft and flabby, its muscular fibres being in a state of fatty and granular degeneration. The voluntary *muscles* are also the subject of a degeneration first described by Zenker in typhoid fever, but now known to occur in typhus and other prolonged febrile conditions. The muscular fibres are converted into a homogeneous, colourless, waxy-looking material, forming cylinders, which break up into fragments, and finally crumble into a granular detritus. This

mesenteric gland

spleen
liver
kidney
heart
muscle

ordinary occupations, until, perhaps, an indiscretion in diet, or the use of aperients, given in ignorance, leads to a fatal perforation. Cases so mild as this in their general symptoms, and yet so dangerous from their possible termination, have been called ambulatory typhoid. A bilious form of typhoid has been described, and in a few cases jaundice occurs. Ataxic and adynamic forms have also been described, but these terms simply indicate the predominance of symptoms in one or other system of the body. Enteric fever is often very mild in children, often of short duration, and associated with less extensive disease of Peyer's patches than in the average of adult cases. The remissions of temperature, which are well marked in the latter half of the illness in adults, are often still more marked in children, and the "infantile remittent fever" of older writers was undoubtedly enteric fever. Haemorrhage & perforation & less also

mild

Bilious

ataxic

adynamic

Children

Diagnosis.—A great number of diseases may be confounded with enteric fever, from the variety of forms which it assumes, and from the frequency with which its own typical symptoms are absent or badly marked. In early stages it is distinguished from the exanthems by the absence of characteristic eruption. By the fifth day of the illness, the rash of typhus, small-pox, or scarlet fever would have developed; the appearance of rose spots a few days later confirms the suspicion of enteric fever. Sometimes severe joint pains may lead to a suspicion of rheumatic fever. A prolonged febrile complaint, which has come on insidiously, and presents no obvious local lesions, should always excite a suspicion of enteric fever; but the great prevalence of influenza gives rise to frequent mistakes. For though influenza is often a much more sudden and quickly prostrating disease, it presents so much variety that almost any illness beginning with headache, backache, and fever is liable to be taken for it. If typhoid fever is present, the temperature remains high, or even rises, and the diagnosis may be soon confirmed by diarrhœa, enlargement of the spleen, or rose spots.

Exanthems

Typhus

Small-pox

Scarlet fever

Rheumatic fever

Influenza

Later stages present a resemblance to different diseases, according as the head, chest, or abdomen shows the most prominent disturbance. Thus, the early headache of typhoid, and the subsequent delirium, may suggest *meningitis*, and the two diseases are frequently confounded together. Sometimes it is impossible to distinguish them until later stages, when optic neuritis or a local paralysis, squinting or convulsion, or the obstinately retracted abdomen may decide for tubercular meningitis; or, on the other hand, the increase of abdominal symptoms, with the presence of spots, may prove it to be enteric fever. In this latter, headache rarely continues beyond the tenth day. When pulmonary symptoms are marked, *acute general tuberculosis* may be simulated by the abundant bronchitic râles and crepitations, accompanied

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Though the reaction is nearly always o
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The test solution, which should be
being used, consists of a concentrated aqu
acid, 200 c.c.; pure nitric acid, 10 c
solution of pure nitrite of sodium, 6 c.c.
alkaline with ammonia; then add an
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twelve or twenty-four hours, it deposi
stratum of which shows a light or dark
colour.

Widal's Serum Test depends upon the
of patients with enteric fev
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the end of the first week, subsequently never above 103 , e is favourable; if the temperature is maintained at 104 ier throughout the second week, it is much more dangerous. ases sink rapidly by the twelfth, eleventh, and tenth days, before this. Perforation is very rarely recovered from. rhage is less dangerous, but a severe hæmorrhage, if it ot lead to a fatal result, renders the patient very anæmic, nsiderably prolongs convalescence. Much abdominal dis-, profuse diarrhœa, severe general bronchitis, and a feeble egular heart are all unfavourable.

tment.—The patient should be in bed in a well-ventilated ent, and the same rules should be carried out as to nursing ie case of typhus fever. The special dangers of perforation morrhage from the ulcerated bowel should never be lost f. Rest should be absolute; the patient should be allowed rtion, and a bed-pan should be used when he wishes to s motions or urine. The diet should consist chiefly of milk, h two, three, or four pints may be given daily, in regular ies, every one or two hours, or more frequently. Beef-tea so be given, but it is said sometimes to increase the ea, and is certainly not as nutritious as milk. In some where milk is taken badly, it may be peptonised by the use ger's liquor pancreaticus. As to medicinal treatment, in ases little or none is wanted. A small dose of dilute l acid, or of a saline diaphoretic like the acetate of iium, may be grateful to the patient, and the body may itly be sponged with tepid water. With regard to the stration of stimulants, the remarks made in the chapter hus apply in the case of enteric fever.

ial symptoms or complications may have to be met, such as itis, by small doses of expectorants; or persistent headache, risk of bed-sores, but also to prevent the possibility of the attendants being infected by the development of the specific poison.

Constipation is not infrequent, especially in the latter half of the illness, after a short diarrhœa in the early days. The bowels may be left for four or five days without harm, and it is then safest to use a soap enema from time to time as required. If there is constipation at the beginning of the illness, in the first week, and before ulceration has begun, a small dose of castor oil may be given. Under no circumstances should the more active or drastic purgatives be employed.

The above is a simply expectant line of treatment, by which the patient is cared for while the disease runs its course. A more effective control over the progress and accidents of the fever has been hoped for by two methods of treatment which may be called respectively (1) the antipyretic; (2) the antiseptic; but it cannot be said that either of them shortens the duration of the disease.

The first, or *antipyretic*, has been in use now for some years. It aims at improving the chances of the patient, and reducing his liability to complications, by lessening the average temperature throughout the illness. The theory of this system is that the continued high temperature, to which the tissues and organs of the body are subjected, is the chief cause of their granular degeneration; and that their ultimate failure, or the occurrence of complications, is so much more probable, the higher the fever. Hence the object should be to bring down the mean temperature, and this can be most conveniently done by reducing the temperature 3° or 4°, at more or less regular intervals. The methods employed have been already described under the general treatment of fevers (p. 25).

As to the action of the cold bath, a good deal of evidence has been accumulated. The influence of a single bath upon the immediate condition of the patient is generally most pronounced. Headache, delirium, stupor, thirst, are at once diminished, the tongue becomes clean, the pulse slower and firmer, and the patient feels altogether relieved. But this effect is only temporary, and by the next observation the temperature may be as high as before. When the system is thoroughly carried out by frequent baths at low temperatures, the mortality has been markedly reduced, and even in its modified forms a decided improvement has been noted. Its effect upon complications is also marked—bronchitis and hypostatic congestion improve, and it has been shown by statistics that hæmorrhage is less frequent. The contra-indications are extreme collapse of the patient, hæmorrhage already established, and severe conditions of pulmonary congestion. These are likely to be avoided by the adopreturns with remarkable slowness, and the patient should not be too early allowed to exert himself. Rarely is one fit for work under three months from the commencement of the illness, and in the graver forms, or in case of relapse, or of complications, this period may be prolonged to five or six months.

MEDITERRANEAN FEVER.

(*Malta Fever.*)

This is a continued fever of long duration, bearing some resemblance to enteric fever, but distinguished from it by the absence of rose spots, the fact that Peyer's patches are not enlarged or ulcerated, and the low mortality. It occurs along the shores of the Mediterranean and among its islands.

Ætiology.—It affects the sexes about equally, but it is more frequent in the young than in the old. It occurs more frequently in the warmer months of the year. It appears likely that it is conveyed by means of drinking-water; and at any rate it is not contagious from person to person directly.

Symptoms.—Its *incubation* period varies from a few days to three or four weeks. The symptoms come on insidiously, and consist of pyrexia, headache, pains in the bones, sleeplessness, thirst, furred tongue, loss of appetite, nausea, and weight and tenderness in the epigastric region. The bowels are usually constipated; the spleen is always enlarged, and often tender or painful; sometimes the liver is enlarged; and profuse perspirations occur with crops of sudamina. The headache and severer symptoms may subside in two or three weeks, but the pyrexia continues for much longer, even up to three months, and only slowly subsides. During this time the patient becomes anæmic. In about half the cases the joints become red, swollen, and painful; and in a few there is orchitis. The temperature appears not to be continuously high over all this time, but to have exacerbations of two or three weeks, with intervals of a much lower or nearly normal temperature, so that a close resemblance to the chart of a relapsing enteric fever is presented. The mortality is only about 2 per cent.

Morbid Anatomy.—The spleen is large, soft, and diffluent; the liver and kidneys are congested, and the latter are in a condition of glomerular nephritis. The mesenteric glands are slightly enlarged. Peyer's patches are to the naked eye normal; the most that can be seen with the microscope is some slight proliferation of cellular elements. D. Bruce discovered in the spleen a micrococcus (*M. melitensis*), which is undoubtedly the specific cause of the disease.

Treatment.—Neither quinine nor salicylates have any influence on this disease. It may be treated in the same way as a mild enteric fever by milk diet; and troublesome symptoms must be met as they arise. For the joint affection the use of iodine, or friction with liniments, is recommended (Bruce).

WEIL'S DISEASE.

The illness described under this name on the Continent is an acute febrile disease, of short duration, accompanied with jaundice and swelling and tenderness of the liver. It is most common in young adult males. As a large proportion of the patients have been butchers, and the disease has occurred in the hot season of the year, it is likely that the disease arises from contact with decomposing animal matter. It begins suddenly, often with a chill, and without prodromal symptoms; the symptoms are fever, headache, signs of gastric disturbance, jaundice, and muscular pains, especially in the calves. The fever lasts eight or ten days. Sometimes there is a relapse. The pulse is quick at first, and afterwards slower than normal. The spleen and liver are commonly, but not always, swollen, and the liver is often tender on pressure. Nephritis is often, and herpes and erythema are occasionally, observed. The prognosis is favourable. The recorded cases are at present not numerous, and the nature of the disease is uncertain. It is not any form of enteric fever, nor relapsing fever, nor catarrhal jaundice: a probable view is that it is the bilious typhoid formerly described by Griesinger in Egypt, and still met with in that part of the world.

SCARLET FEVER.

(*Scarlatina.*)

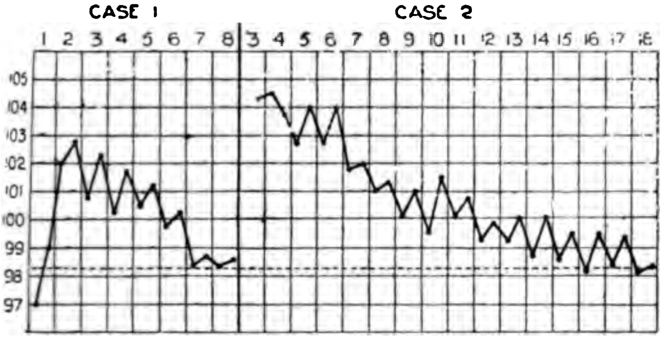
Scarlet fever is a contagious disease, characterised by fever, sore throat, a bright red eruption on the skin, and a tendency to certain complications, of which the most important is acute inflammation of the kidneys.

Ætiology.—Scarlatina is invariably derived from a preceding case of the disease, either by direct contagion from the breath or exhalations of the sufferer, or by minute particles of shed epithelium, or by dried secretions from mucous surfaces. It is much more readily conveyed to a distance than typhus or relapsing fever, and it is remarkable for the tenacity with which it adheres to clothing, bed-clothes, books, papers, and other articles that have been used by the sick; so that the contagium has been conveyed by such means over miles of country, or has lain dormant for weeks or months, and then, meeting with a suitable nidus, has again developed the disease in its complete form. Milk may, as in typhoid fever, be a means of conveying infection, which has in some instances probably been derived from a similar disease in the cow. Sex, occupation, and social position have no influence in its production, but the vast majority of those attacked by scarlatina are young children, and it is comparatively infrequent in adults. This exemption of older people is explained by the fact that, for the most part, one attack protects against future attacks, and that most adults have already had the disease. Still, old people occasionally have it; and protection is not always perfect, so that some people have a second attack. It has also been noticed that very young infants are less susceptible than older children.

Symptoms and Course.—The period of *incubation* is two, three, or four days, rarely as much as six days. Generally the invasion is sudden: the patient has a rigor, or vomits, and complains of frontal headache, with languor, pains in the back and limbs, and loss of appetite. The temperature rises to 103° or 104°, the pulse becomes very rapid, and the respiration is quickened. Very soon there is some complaint of sore throat, and swallowing is painful.

On the second day—that is, generally between twelve and thirty six hours from the first symptom—the *rash* appears. It is first seen on the upper part of the chest, in front and on the sides of the neck, but soon spreads to the abdomen and back, and then to the upper and lower limbs. It consists of minute red spots, bright in the centre, fading towards the edge, and set closely together, so that the paler edges almost coalesce. Sometimes the coalescence is complete, so that the skin has a uniform bright red colour; sometimes the eruption is more discrete, and areas of pale skin are visible between the spots. The face, forehead, and cheeks are as a rule deeply and uniformly flushed, without showing the punctiform arrangement of the rash which is seen elsewhere. With an abundant rash the skin becomes slightly swollen. The eruption presents many varieties as to depth of colour and distribution. It may be only pale pink, or it is deep livid purple; and in some severe cases papules may be raised above the surface, and may even vesicate or form minute points of pus; and occasionally petechiae occur. In its distribution the rash may be very limited, occurring only on the chest, or in patches on the thighs, elbows, or ankles, and this occurs frequently in second attacks, and in the mild cases sometimes seen in patients with open wounds, and known as "surgical scarlatina." The rash reaches its height on the third or fourth day, and begins to fade on the fourth, fifth, or sixth day; altogether it may last from five to ten days. After the subsidence of the rash, *desquamation* takes place—that is, the superficial layers of the cutis are shed. This is often first seen in the form of white branny flakes on the sides of the neck, and this may be as early as the sixth or seventh day, while the eruption is still visible on the legs; but the amount of epithelium that is shed, and the size of the particles, are very various—sometimes there is nothing more than a little roughness about the tips of the fingers or toes, or in the folds of the palms of the hands; while in other cases the epidermis peels off in large flakes, and, in rare instances, complete glove-like moulds of the hands and fingers are thrown off. Desquamation commonly takes from four to six weeks, but in these special cases a much longer time is required.

Fig. 6.



Temperature in Scarlatina.

In the throat it is seen that the uvula, soft palate, and fauces are deep red, and often slightly œdematous; the tonsils are reddened, swollen, projecting towards the middle line, and presenting a number of yellow points, from the follicles being distended or covered irregularly with ashy or yellowish secretion. In later stages they may suppurate, or sloughs may form in them. The nasal mucous membrane also inflames, with the occurrence of much mucous secretion; and the submaxillary and cervical glands become enlarged and tender. The tongue is at first thickly covered with white fur, but in a few days this fur clears off from tip to base, leaving a bright red, raw surface, on which the fungiform papillæ are unusually prominent, so as to give the appearance known as "strawberry tongue."

The temperature is frequently high, reaching 104° or 105° on the

Temperature

Pulse

Delirium at night

[illegible]

[illegible]

first day, and remaining at this level for some days. Even a temperature of 106° may be reached. With this the skin is pungently hot and generally dry, but profuse sweating may occur, without appreciably affecting the temperature or the rash. The pulse rises to 120, 140, or even 160. In severe cases, the mental faculties are dulled; delirium is frequent, especially towards night; and drowsiness and coma supervene. The disease may reach its height about the fourth, fifth, or sixth day, and then with the fading of the rash, the temperature begins to fall, generally subsiding rather gradually, but sometimes more suddenly, till the normal is reached, and convalescence is gradually established. In fatal cases, death may occur about the fifth day or later from exhaustion; or from typhoid conditions, with low delirium, semi-coma, and dry brown tongue; or it may occur later still as a result of complications.

Tonsils, Uvula

Glands

Otitis

[illegible]

Deafness

[illegible]

Later (Brain, Mastoid)

Meningitis

Abscess of [illegible]

Pyaemia

Haemorrhage

Facial Paralysis

Deafness

Cornea

Sloughing of

Abscess

Cancrum Oris

Nephritis

Complications and Sequelæ.—These are numerous and important. Not only the tonsils, but also the soft palate and the uvula may slough. More frequently the glands under the jaw and in the neck are much swollen, and the subcutaneous tissue about them is infiltrated, becoming brawny and indurated. The skin then becomes dusky red, and sloughing takes place beneath it, separating it from the subjacent tissues over a large area. Such cases are often fatal. Extension of the inflammation from the throat up the Eustachian tube may cause *otitis*, or inflammation of the ear, resulting in abscess of the tympanum, rupture of the membrana tympani, and otorrhœa. In the course of the scarlatina this may seem of little importance; but it lays the foundation for serious or fatal results months and even years afterwards, among which may be enumerated suppuration of the mastoid cells, meningitis, abscess of the brain, thrombosis of the lateral sinus or jugular vein with pyæmia as a result, hæmorrhage from the lateral sinus, and facial paralysis. Deafness on the affected side may of course happen; and a double otitis in a young child may be the cause of permanent deaf-mutism. Other local lesions may occur as sequelæ; for instance, sloughing of the cornea, abscesses in the subcutaneous tissues, or cancrum oris.

The most important visceral lesion associated with scarlatina is *nephritis*. It is still a question for discussion whether the kidney is affected in every case of scarlatina; but albuminuria is occasionally present in the stage of rash and fever. More frequently nephritis is first recognised as a sequela from two to three or four weeks after the beginning of the illness, when the patient is quite convalescent or suffering only from desquamation. It may begin with a chill and rise of temperature, and the passage of turbid brown or blood-coloured albuminous urine, the whole subsiding again without the occurrence of dropsy; but more often the first

Tonsils

the bases of the lungs. The tonsils present the conditions of ulceration or suppuration that have been observed during life. Where complications are present, the visceral lesions proper to them will be found.

Infection

Streptococci have been discovered in the blood, in the scales of the desquamating epidermis, in the ulcerations of the throat, and in the discharges from it; but their specific nature is not yet allowed.

Measles
Rubella
Typhus
Small Pox

Diagnosis. — Scarlatina is recognised, especially when the disease is known to be prevalent at the time, by the occurrence of feverishness with sore throat, followed in a day by the characteristic rash. The rash may be confounded with that of measles, rubella, or typhus, or with the roseola of small-pox and other fevers. Generally, it is of brighter colour and more uniform distribution than those of the first three diseases: but it is sometimes very difficult to distinguish from rubella. The roseola of small-pox often has a distribution which is distinctive—namely, about the axilla, groins, and thighs; and it is more purple in colour. Occasionally, confirmation only comes with desquamation and the appearance of albumen in the urine.

Renal
Complications
Confinement
Severe Symptoms

The **Prognosis** must be in all cases very uncertain. Even in the mildest cases, renal complications may be serious or fatal. The mortality, however, is variable, some epidemics being exceedingly mild, others so severe that the mortality may rise to 30 or 40 per cent. In individual cases, the prognosis may have to be determined by the condition of the patient from day to day; the addition of complications increases the danger. Very severe angina; and an intense or livid rash coming out late, are unfavourable; and cases with sloughing of the cervical glands are generally fatal. Scarlet fever attacking women recently confined shows a large percentage of fatal cases.

[illegible]
Air
Rest
Diet
Nursing
Isolation
Sponge
Saline
Ice
Sore throat
Nose
Swelling

Treatment.—In mild cases this may be carried out in the same way as that of other fevers: a well-ventilated room, the recumbent position in bed, light diet, and careful nursing. Isolation is essential in the interests of others. The body may be usefully sponged with tepid water daily; simple salines may be given internally, and the sore throat may be relieved by sucking lumps of ice. If the tonsils are much swollen, and much covered with secretion, the latter should be removed by pledgets of moist lint, and disinfectant or astringent solutions applied by the same means. Liquor ferri perchloridi (ʒss to aq. ℥j), dilute hydrochloric acid (ʒss to ℥j), or liquor sodæ chlorinatæ may be thus used; and similar remedies, more diluted, may be used to syringe the nose when that is involved. If there is much pain or swelling in the neck and about the angles of the jaws, hot fomentations or linseed poultices should be used. Complications will require special treatment. Abscesses should be opened early. In otitis, of pain may be obtained by hot fomentations, and the gentle luction of warm water into the meatus. If suppuration of iiddle ear is recognised, the membrana tympani may require ure; and the meatus may be gently syringed with warm or diluted Condy's fluid, or solution of boric acid (1 in 20). ynovitis, small doses of Dover's powder, or, better, salicylate ium should be used, with chloroform or belladonna liniments . The treatment of scarlatinal nephritis will be considered ter. In the severe typhoid forms, with quick feeble pulse, ants, such as brandy and ammonia, must be given; and there is very high fever, with much delirium and restless- relief is often obtained by cold affusion to the head or

at care must be taken during convalescence, both for the f the patient and in the interests of the public. To the t the chief danger is the supervention of renal complications: at should be guarded against by avoiding every exposure l or draught as long as desquamation is going on: a free of the bowels should also be maintained by occasional saline 'es. On the other hand, the poison of scarlatina is con- by the particles of detached skin, and the disease is fre- y spread by the careless exposure of convalescents in the of desquamation. Patients should not be allowed to mix he healthy until desquamation is complete, and six weeks en adopted as the minimum period at which they may be ered free from risk to others. During free desquamation, dy should be washed night and morning with soap and water, and gentle friction with pumice-stone may be ed to remove the desquamating epithelium; and in the ls the body should be smeared with carbolic oil (1 to 40) glycerin to prevent the particles being carried off into the here. Quinine and other tonics may be necessary where th is recovered slowly.

MEASLES.

(*Morbilli.*)

sles is a contagious febrile disease, characterised by an n of pink or red spots, and catarrh of the respiratory s membranes.

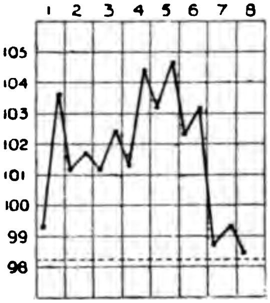
ology.—In civilised communities its spread is determined umstances very similar to those influencing scarlatina. It in epidemics, which attack the young rather than the old, because nearly all the older members of the community have had the disease when young, and are thereby protected from a second infection; but very young infants appear to be less susceptible, and to have the disease, when caught, in a milder form than children somewhat older. In large towns it is almost continuously present, spreading from point to point in the form of limited outbreaks, which subside, and are succeeded by others in different places; but where introduced among populations that have never been visited by the disease, or have been entirely free from it for years, it attacks the majority of the people, young and old alike, in one great and often destructive epidemic. This was the case in the Faröe islands in 1846.

Infection is mostly produced by contact with the patient, or with the air infected by him: the virus also adheres to clothes and other articles, though with much less tenacity than does the contagion of scarlatina. Experimentally also, the disease has been transmitted by inoculation with the nasal mucus of a patient. It is especially contagious in the prodromal stage preceding the rash, and in the stage of eruption itself.

Symptoms and Course.—The period of *incubation* varies in measles as in other contagious diseases, but has been most often found to be about ten days. The disease commences with pyrexia and catarrhal symptoms—the temperature rises perhaps to 102°; the child loses appetite, is drowsy, and unwell; there may be, at first, vomiting or chills, or, in children, convulsions. With this the conjunctivæ become suffused, the eyes water, there is a mucous discharge from the nose, and cough as a result of bronchial secretion. The catarrhal symptoms continue, but the temperature frequently falls after the first day, and continues at a lower level for another day or two, when it again rises with the outbreak of the rash. This appears most commonly on the fourth day, but it may be as early as the third. It is first seen on the face, at the roots of the hair, on the forehead, temple, or behind the ears, and it subsequently spreads to the neck, trunk, and limbs. It consists of pink spots, round, oval, or irregularly shaped, slightly raised above the surface, running together into irregular patches, which may have a somewhat crescentic shape, and leave some intervening area of skin unaffected. In colour, it is generally darker red, or more purple than that of scarlatina; but a distinction may be difficult, especially if the spots are uniformly distributed and do not coalesce. Occasionally, a few petechiæ occur in the darkest part of the eruption, and in other cases a few vesicles may form in the centres of some papules.

It comes out most fully on the face, giving it a blotchy, swollen appearance, and is less abundant and less confluent on the trunk and extremities. It takes from one to three days to reach its height, and then rapidly declines, mostly beginning to fade first where it first appeared. It commonly leaves some mottling of brown or yellowish-brown colour, which lasts for some days; while petechiæ will leave still more pronounced stains. It is also succeeded by slight desquamation in minute branny scales, never in the large flakes known in scarlatina. If the temperature has fallen in the prodromal stage, it rises again, with the appearance of the rash, to 102°, 103°, or even higher, and reaching a maximum in two, three, or four days, falls generally rather suddenly as the rash begins to fade, and may reach the normal in about thirty-six hours. The catarrh continues throughout the eruptive stage; it may extend into the frontal sinuses and cause headache. There is more or less general bronchitis, indicated by cough, expectoration of mucus, and diffused rhonchi; and the larynx may be implicated, as shown by hoarseness, croupy cough, and in occasional cases by stridulous breathing. Early in the illness, before the appearance of the rash, the palate often shows abnormal redness, diffused or in patches. The tongue is usually furred, and the fungiform papillæ are prominent. After the subsidence of the fever the return to normal appetite and sleep is generally rapid.

FIG. 7.



Temperature in Measles. (*After Strümpell.*)

Complications and Sequelæ.—The most important complications are those connected with the respiratory organs, and it is to them that the majority of the deaths in connection with measles are due. The bronchitis of the large and medium-sized tubes, which is common to all cases of the disease, may attack the smallest tubes, thus becoming a capillary bronchitis; or the substance of the lung may be involved, constituting pneumonia. This may present the characteristics of catarrhal or broncho-pneumonia, but it is not infrequently limited to one lobe, with dulness, bronchial breathing, and consonant râles similar to cases of ordinary or croupous pneumonia. Thomas, indeed, states that the pneumonia following measles may be either of the catarrhal or croupous variety; and it has been observed that the organisms of broncho-pneumonia are present with unusual frequency in the mouths of children with measles. The laryngitis may be so severe as to threaten asphyxia, and may be accompanied with the formation of membrane; in some such cases it is a true diphtheria; in others it is caused by pyogenic organisms. Other complications are ophthalmia, keratitis, or iritis, stomatitis and parotitis, inflammation of the Eustachian tube, and of the tympanum (*otitis media*), and diarrhœa or dysentery from enteritis of the small or large intestine respectively. Noma, or gangrenous inflammation of the mouth or of the vulva, occurs occasionally. Amongst sequelæ may be mentioned, especially, chronic inflammatory conditions of parts affected during the illness, such as chronic catarrh of the respiratory organs, chronic ophthalmia, enlarged tonsils, or enlarged glands, otitis with discharge and its results (*see* p. 56), tuberculosis, endocarditis, and general malnutrition.

Varieties.—Measles without eruption and measles without catarrh have both been described; but there seems to be some doubt whether the former occurs, and some cases of the latter may really have been instances of rubella or German measles (p. 64). In either case, the disease takes on a mild form. Of the severer or malignant forms, *hæmorrhagic* measles is an example, in which bleeding takes place from the mucous membranes, and the eruption becomes *purpuric*. Other severe forms are merely characterised by intense fever, dark or livid rash, often imperfectly developed, rapid and feeble pulse, prostration, delirium, dry brown tongue, and a generally typhoid condition.

Morbid Anatomy.—This must depend much on the complication causing death, since uncomplicated measles is so rarely fatal. Redness of the mucous membrane of the larynx and trachea is observed, the spleen is moderately swollen. No special description is needed of the bronchitis and pneumonia found in cases dying therefrom. There may be, however, fluid in the pleural cavity, and petechiæ under the pleural membrane; the bronchial glands are often enlarged and softened. Congestion of the mucous membrane of the ileum and colon is also sometimes found.

No micro-organism has been identified as the certain cause of measles.

Diagnosis. A confusion with scarlatina is most likely to occur. Measles is distinguished by the initial coryza, by the longer prodromal period, and the character of the eruption, especially in its patchy appearance, as distinguished from the uniform or finely punctate distribution in scarlatina. But the throat may be sore in measles, and the rash sometimes appears on the third day, so as to make the resemblance to scarlatina rather close. Measles may also be mistaken for rubella, in which the fever is slight or none, the prodromal period short or absent, catarrhal symptoms wanting, the rash in smaller patches, more uniformly distributed without crescentic arrangement, the liability to complications very small, and a fatal termination unknown. In typhus fever the rash is not so papular, the face is but little affected, the spleen is swollen, and there is no nasal or conjunctival catarrh. The early stage of a small-pox eruption is sometimes simulated by that of measles; the absence of catarrh, and the history of headache, back-pain and sickness, are in favour of variola. Roseolous eruptions, apart from specific fevers, may resemble measles, but will be distinguished by the absence of the characteristic fever and catarrh.

Prognosis.—This is usually favourable. For the most part, the mortality is from 1 to 2 per cent., though occasionally epidemics of much greater severity have occurred. As already stated, it is the pulmonary complications that are generally fatal; apart from these, the malignant cases are recognised by intense fever, dark or livid eruptions, and early collapse or prostration.

Treatment.—The treatment of measles is not essentially different from that of scarlatina. The child must be placed in a suitable room—warm, well ventilated, and free from draughts, and so arranged as to prevent infection of children hitherto free. Confinement to bed is scarcely necessary till the eruption appears. The diet, as in other febrile affections, must be mainly milk, with a little farinaceous food. The catarrh which is present from the first should be treated by expectorants, squills, or ipecacuanha wine, or small doses of compound tincture of camphor. Goodhart recommends the application of glycerin, or borax and glycerin, to the fauces and throat to relieve the harder kinds of cough. If fever runs high, it may be reduced by the application of tepid water, either by sponging or by immersion in a bath at 95° or 90°, which may be lowered still more by pouring in cold water. The temperature seldom remains at a high level, such as 105°, for many hours, but it may be desirable to spare the patient this prejudicial condition as much as possible; and even at a temperature of 102° or 103° a good deal of comfort and even sleep may be obtained by the use of cold sponging, as has been shown when treating of enteric fever. Stimulants are only required in the very severe forms. The child may generally be allowed to get up two or three days after the subsidence of the fever, but should be confined to the room for another week or ten days. During convalescence the general health should be attended to, and iron or cod-liver oil may be given, with, perhaps, change of air to the seaside or other bracing locality. Pneumonia or diarrhœa may be treated in the usual way, and discharge from the ears should be met by frequent washing with antiseptic lotions—*e.g.*, potassium permanganate or boric acid.

RUBELLA.

(*Rubeola, Rötheln, German Measles.*)

The nature of this contagious exanthem has been much debated. By some it has been spoken of as a hybrid of scarlatina and measles; by many more it has been regarded as simply a mild form of ordinary measles. But there can be now no doubt that it is a distinct disease. Those who have had measles are not protected against rubella, nor those who have had rubella against measles. There is more than one instance on record where a school has been visited by an epidemic of measles shortly after one of rubella, or *vice versâ*: the outbreaks have been obviously different, and some boys have caught both complaints.

Ætiology.—Rubella is very contagious, exposure to the surroundings of a patient for a few minutes being sometimes sufficient for infection.

Symptoms.—The period of *incubation* is probably about ten days, but may be as long as twenty. A prodromal stage may be entirely absent, or at most last half a day, before the appearance of the eruption; and this stage may be represented by a slight catarrh of the mucous membranes of the air-passages or of the conjunctiva. But in some cases the *eruption* is the first indication of anything wrong with the patient. It consists of a number of pink spots, round or oval, very slightly raised above the surface, uniformly scattered, and generally discrete, though sometimes very closely set. The spots vary in size: when small and closely set there may be much resemblance to a scarlatinal rash; when larger there is more likeness to measles, but they are not commonly confluent, and do not take any crescentic form. Slight itching of the skin may be experienced. The eruption occupies the face, trunk, arms, and legs, appearing mostly on the face first, and rapidly occurring on the other parts; it is generally of shorter duration than measles, often lasting only two days, sometimes three or four. As in measles, it may leave a little discoloration of the skin for some days afterwards; desquamation is commonly absent, and it is never in large flakes, as in scarlatina. The palate and fauces usually show some injection or spots and streaks of redness, and the tonsils may be a little swollen. The conjunctivæ are reddened, and coughing and sneezing are generally present to a slight extent. The *lymphatic glands* at the back of the neck are frequently swollen, and sometimes those in other parts of the body. The swelling may persist two or three weeks, but suppuration has never been observed. Fever is, in the majority of cases, entirely absent; if it occurs, the temperature is only 1·5° or 2° above the normal, and lasts one, two, or at most three days, showing the greatest variability in different cases, but often falling to normal before the eruption is completely developed. Many patients do not feel ill at all, and retain their appetite throughout. Any further complications than those indicated already are quite uncommon, and the prognosis is exceedingly favourable.

Glands

Fever

Diagnosis.—This has to be made from measles and from scarlatina. As against the former, note the absence or shortness of the prodromal stage; the slightness of the fever and of the catarrhal symptoms; the round or oval shape, with smaller size and paler colour of the spots, which do not run together into extensive or irregular confluent patches; the rapid extension of the eruption, or its outbreak over the whole body at the same time. From irregular and mild forms of scarlatina it must be distinguished by the larger size of the spots and their more scattered distribution.

Measles

Scarlatina

Treatment.—This must be conducted on the lines laid down for measles.

SMALL-POX.

(*Variola.*)

Small-pox is a specific contagious disease, with a characteristic pustular eruption.

Ætiology.—This disease arises solely by contagion—chiefly, no doubt, by inhalation of the atmosphere surrounding infected persons; but it is also conveyed by clothes, bedding, and other things, which have been in contact with patients. Further, the disease can be inoculated by means of the contents of the pustules—a proceeding which was made use of before the introduction of vaccination, because the disease thereby produced was generally of a milder type, and thus protected the individual from the risk of a more serious attack. But patients are infectious also before the eruption, and the virus is given off even from the bodies of those who have died. The susceptibility to the disease is common to all ages and both sexes; even the fœtus *in utero* may catch it from the mother; but the susceptibility then and in the first year of life is stated to be less than afterwards. Negroes are said to be more liable than white people. In the present century the liability to the disease, and consequently the number and severity of its epidemics, have been reduced considerably by the process of vaccination. The disease commonly occurs only once in the same individual; but second and third attacks occasionally occur, and the second attack may even be more severe than the first, though it is generally milder.

Symptoms and Course.—The period of *incubation* is, in a large proportion of cases, about twelve days, during which, as a rule, the patient is well. The disease mostly begins suddenly with a distinct rigor, or chills, with pains in the back—that is, the loins and sacrum—severe headache and vomiting. The temperature rises rapidly to 102°, 103°, or 104°, and the next day it may be still higher. The patient is mostly very ill, unable to continue his work, and probably takes to his bed. Anorexia, thirst, furred tongue, and constipation are also present. On the third day the typical eruption appears; but in the initial stage in a certain number of cases rashes occur, with which it is important for diagnostic purposes to be acquainted.

Early Eruptions.—These are either *erythematous* or *hæmorrhagic*. Of the erythematous rashes, some cover the whole body and face, and either closely resemble scarlatina or are more like measles; in other cases the rash is partial in its distribution, and has been especially noted on the external surfaces of the arms and legs. Of the hæmorrhagic rashes the most characteristic is one which occupies the lower half of the abdomen, from the umbilicus downwards, covers the groins, and extends on to the thighs in a triangular form, with the apex downwards, corresponding closely to Scarpa's triangle (crural triangle of Simon); it also frequently appears in the axillæ, and on the adjacent parts of the arms and trunk, and extends thence along the flanks to the lower patches. It consists of small hæmorrhagic spots, or petechiæ, which, on fading, leave brown or yellowish-brown stains for a time. These initial rashes commonly appear on the second day, and last for about two days, co-existing, perhaps, with the early stage of the pustular eruption, but disappearing before its full development.

Another form in which an initial rash appears is that of the *purpura variolosa*, which constitutes a very severe variety of the disease. On the second day, or even within twenty-four hours of the first symptom, a scarlatiniform rash appears, quickly followed by subcutaneous hæmorrhage, partly petechial, partly in larger patches. The face is red and puffy, the eyes suffused; there may be vomiting of bilious matters or of blood, with the passage of bloody stools, and the urine contains albumen or blood. The mind is generally clear till near the end; exceptionally, delirium or coma is observed. The cases are nearly always fatal, often within three days of the commencement, and even earlier.

up the swelling of the face and other parts subsides. Finally, the scab falls off, leaving a deep red spot, which is at first slightly raised above the general surface; but in the course of some weeks forms a depressed white scar. The pustules form most abundantly on the face, and on the backs of the hands, and are less numerous on the trunk and covered parts of the limbs. Parts that have been the seat of initial erythematous or petechial rashes are by many said to be less liable to the specific eruption: but Curschmann thinks the comparative exemption of such parts is probably independent of this accident. All the stages of the eruption occur first on the face, and follow, a day or so later, on the trunk and extremities. On the other hand, parts which have been irritated, as, *e.g.*, by the application of plasters or blisters, are liable to an abundant formation of pustules. The pustules are not confined to the skin, but occur on the mucous membranes also; they are especially well seen in the mouth, on the hard and soft palate, but present different appearances from those on the skin on account of the constant moisture to which they are subject. They scarcely develop into well-formed pustules; but are only greyish or pearly elevations, which are liable to become abraded and form superficial erosions or ulcers. The tongue is generally coated and more or less covered with pustules; rarely its substance is inflamed. Ulceration of the larynx or even perichondritis may occur; and the process may extend to the nasal mucous membrane, so that the breathing is obstructed by the swelling and the formation of scabs.

General Condition.—In mild cases the *primary fever* falls with the appearance of the specific eruption, and the temperature may be quite normal; so that patients who have remained at home during the first three days will go to their doctor or the hospital with an abundant crop of papules all over the body, but feeling comparatively well, free not only of the fever, but of the headache, lumbar pains, vomiting, and general malaise.

But when the pocks become purulent a fresh access of fever takes place, a *secondary* or *suppurative fever*, which may be ushered in by chills or a rigor, and lasts from three to six or eight days. The temperature rises to 103° or 104°, is mostly remittent in its course, and is accompanied with sleeplessness, headache, and delirium, and a pulse of 100 to 120. All this again subsides as the scabs dry, and the swelling of the skin decreases. With the fall of the scabs some patients lose their hair, and even their nails.

Varieties.—Perhaps no disease has been honoured by so much subdivision as small-pox, numerous epithets having been devised to distinguish differences in the eruption, besides such as have reference to the general course of the symptoms. It is not necessary to describe all these: it will be sufficient to speak of the following varieties:—(*a*) ordinary or discrete small-pox, to which the previous description mainly applies; (*b*) confluent small-pox; (*c*) malignant small-pox, the purpura variolosa before mentioned; (*d*) hæmorrhagic small-pox (*variola hæmorrhagica pustulosa*); (*e*) modified small-pox; and (*f*) inoculated small-pox.

(*b*) *Confluent small-pox* is a form in which the eruption is very abundant, and the general illness is correspondingly severe. The initial fever is high, and the temperature does not fall to normal with the appearance of the rash, as it does in mild cases. The rash appears early, even by the second day, and is very abundant, so that on the face, which is most affected, the pustules are closely set, the skin is enormously swollen, and in the stage of suppuration several pustules coalesce and form irregular and more or less extensive purulent blebs. The implication of the mucous membranes of the nose, throat, and larynx is much more constant and severe. The secondary fever is also high, and is accompanied by prostration, rapid pulse, and delirium or coma. Complications are more frequent and severe and the mortality is great, death taking place from exhaustion, or hyperpyrexia, or pyæmia.

(*c*) *Malignant small-pox*, or purpura variolosa, in which hæmorrhages appear in the skin within the first forty-eight hours, has been mentioned (p. 66); in another form (*d*) the hæmorrhagic tendency shows itself later. The specific eruption appears, and then hæmorrhage takes place into the papules, or later still into the pustules, or into the skin between the pustules. The petechiæ often occur first on the lower extremities. The mucous membranes are also affected with hæmorrhages, or diphtheritic patches, and bleeding occurs from the nose, lungs, rectum, kidneys, or uterus. The cases are mostly fatal.

(*e*) *Modified small-pox*, often called *varioloid*, occurs for the most part in those who have been vaccinated, but in whom the protection is incomplete, either from deficient vaccination originally, or from the decline of its influence with lapse of time. No broad line of distinction can be drawn between this and the milder forms of discrete variola, but it presents many irregularities. It may be altogether milder, with less fever, and fewer papules; and the eruption may not go beyond the vesicular stage, or even the papular stage. Sometimes the initial fever is severe, with a high temperature, but it is generally of short duration, the secondary fever is very slight, and the eruption is only imperfectly developed. According to some writers, the erythematous initial eruptions are almost confined to these modified cases. Recovery is the rule.

(*f*) When small-pox is *inoculated* a pimple rises on the second day at the seat of the operation; this develops into a vesicle or pustule. The patient then has rigors, swelling in the axillary glands, and some fever; and about the eleventh day the usual eruption of small-pox appears, and passes through its stages. The attack is generally mild, but it is contagious and occasionally fatal.

The **Complications and Sequelæ** are chiefly as follows:—Abscesses and erysipelas, conjunctivitis, and sometimes destruction of the eye from suppurative keratitis; chronic otitis and caries of the bones of the ear; in the lungs, bronchitis, bronchopneumonia, and pleurisy. Changes in the larynx have been described above. Paralysis of the extremities and bladder also occur, which Westphal has shown to be due to disseminated myelitis.

Pathology.—Apart from the anatomical characters belonging to the above complications there is no more to be found in cases dead of small-pox than in other eruptive fevers. In hæmorrhagic cases blood may be found effused into the solid viscera. Examination of the pustules shows that the process begins with hyperæmia of the papillary layer of the cutis, then the superficial layer of the cuticle is raised from the deeper layers to form a vesicle. The umbilication is sometimes determined by a hair, or the duct of a sweat gland preventing distension at this spot, or merely by cells of the rete Malphigi stretched into a fibre; bands and fibres formed in the same way constitute the septa dividing the vesicle into *loculi*. The pustule becomes hemispherical, in the later stages of suppuration, by the central band or *retinaculum* giving way. Whether the resulting scars are superficial or deep depends upon the extent to which the suppurative process involves the papillary layer of the skin.

Although micrococci have been found in the pus and in various organs, nothing is as yet known of the specific micro-organism of this disease.

Diagnosis.—During epidemics of small-pox the occurrence of shivering, with severe pain in the head and back and vomiting, should make one suspect this disease, but at other times the onset may suggest other acute diseases. The petechial eruptions on the lower parts of the abdomen and in the groins are very characteristic; but the erythematous eruptions may closely simulate scarlatina or measles. On the other hand, measles is the disease most often called small-pox by mistake; and syphilis sometimes presents an eruption extremely like it. The variolous eruption is characterised by its hard papules, appearing first on the face, and the vesicles are often grouped in twos or threes. The special features of purpura variolosa, the early appearance of the hæmorrhages, and possible death before the papular eruption, should be borne in mind; indeed, a severe hæmorrhagic eruption, coming on rapidly, with the indications of an acute fever, is generally due to small-pox.

The **Prognosis** has been already indicated. In modified small-pox and the discrete variety it is favourable; in confluent and hæmorrhagic cases unfavourable. Thorough vaccination and re-vaccination diminish to a remarkable extent the severity of the disease; and accordingly prognosis is favourable in proportion to the evidence that these operations have been successfully conducted—*i.e.*, the number and possibly the size of the scars.

Treatment.—The general lines of treatment are the same as for the infectious diseases already described. The patient must be isolated for the sake of others; he must be in bed in a well-ventilated room, and should have abundance of milk and beef-tea, and cooling drinks to quench thirst.

The surface of the body should be sponged with tepid water, and vaseline may be applied where itching is troublesome. The swelling of the face may be relieved by cold compresses, and the eyelids should be frequently washed, and a little astringent lotion dropped within them. Opium may be given to procure sleep.

In the severer cases stimulants may have to be given freely. Many attempts have been made to prevent the scarring or "pitting," which causes so much disfigurement after a severe attack. Painting the face with iodine, evacuation of the vesicles, and touching with a point of solid silver nitrate, anointing with oil, or carbolised oil, have all been recommended; but it is doubtful if any of the processes is to be relied upon. Dark Room

If an unvaccinated person has incurred the risk of small-pox he should be vaccinated at once, as it is certain that the disease may be favourably modified by this procedure.

VACCINATION.

Prevention of Small-pox.—*Inoculation and Vaccination.*—The observation that small-pox, when conveyed by inoculation of the contents of the vesicle under the skin, produced a milder attack than that commonly conveyed by contagion, led to the use of inoculation as a means of protecting the individual from the more dangerous forms of the disease. Lady Mary Wortley Montagu introduced the custom into England in the early part of the eighteenth century, and her example was widely followed. But a serious disadvantage attached to this proceeding; the small-pox induced by inoculation, though mild, was contagious, and the spread of the disease was thus decidedly favoured. Inoculation consequently fell into disrepute, and finally yielded to vaccination—*i.e.*, the inoculation of *cow-pox*, or *vaccinia*—first practised by Jenner in 1796. He was led to make the experiment from the facts, long observed in dairy farms, that cows were liable to a pustular disease of the udders and teats, which was often accidentally communicated to men and women milking them, and that these persons were subsequently insusceptible to small-pox, either by contagion or by the inoculation then in vogue. Conversely, it was observed that those who had had small-pox did not catch the disease from the cows. Jenner inoculated patients with cow-pox, which produced its characteristic effects, and he subsequently found that certain of these patients were insusceptible to a small-pox virus, which set up typical variola in other unvaccinated persons. The power of vaccination to protect from small-pox has been abundantly proved since then by facts which I need not here discuss.

The cow-pox may be conveyed from man to man by means of the lymph contained in the vesicles a great many times without any very obvious diminution of its power to reproduce the disease and to protect from variola; but that such a diminution does eventually take place is believed by many.

Vaccination in Man.—When cow-pox, taken from the disease in man, is inoculated again under the human skin, nothing occurs till the end of the second or the third day, when a papule appears at the seat of inoculation. This increases in size, and on the fourth or fifth day a vesicle forms, which enlarges and forms a circular bleb, flat, or slightly depressed in the centre, and greyish in colour. On the eighth or ninth day the contents begin to be purulent, and a pink zone of inflammation forms around it. The vesicle becomes more opaque; the redness increases in extent, and is accompanied by induration. The neighbouring lymphatic glands become swollen and tender, and a slight degree of fever and malaise are present at this time. About the tenth or eleventh day the pustule begins to dry, and a brown scab forms during the next few days. The surrounding inflammation subsides, and about the end of the third week the crust falls off, leaving a depressed, pitted, and permanent scar.

The Operation of Vaccination.—The English law requires that all children shall be vaccinated before the age of three months, unless the parents provide a medical certificate that the child is unfit to undergo the operation. It is now proposed to extend this period to six or twelve months. It has been customary to inoculate with lymph taken from another subject on the eighth day after vaccination. The vesicle was punctured and the lymph stored by being allowed to dry on the points of lancets, or on small ivory points, or by being drawn into capillary tubes, of which the ends were at once closed with sealing-wax. But, partly the difficulty of supply from human sources, partly the objections as to the possible transmission or conveyance of disease in human lymphs have led to the use of lymph obtained from vaccination in the calf; and it is claimed that calf-lymph gives quite as certain results in infants, more certain results in re-vaccinated adults, a fifteen years. It thus becomes desirable that every one should be again vaccinated in childhood or early puberty, and subsequently at any age, if small-pox should become epidemic. According to the extent to which the influence has faded, re-vaccination will have different results. It may fail entirely, or only produce a little local irritation; or it may produce a typical vesicle.

CHICKEN-POX.

(*Varicella.*)

Chicken-pox is a specific infectious disease characterised by an eruption of vesicles. Although it has been often confounded with small-pox, there can be now no doubt that it is a different disease. Its occurrence is not affected by vaccination, and it cannot be inoculated. Its specific micro-organism has not yet been discovered.

age

It commonly occurs in children, being conveyed by the air or by clothes; and it very rarely attacks adults.

Incubation

The period of *incubation* is more commonly fourteen days, but may be as short as twelve, or as long as nineteen.

Eruption
Papule
Fluid
Scab

The eruption consists, at first, of pink spots or papules, on which, in twelve or twenty-four hours, vesicles form. These are generally tense, hemispherical, and from one-eighth to a quarter of an inch in diameter. At first the fluid is clear and colourless, but it soon becomes opalescent or milky, and then the vesicle shrivels, and a yellowish or brown scab forms, which adheres for a few days, and then separates, leaving a pink stain. The perfectly formed vesicle is surrounded by an inflammatory zone, which subsides as the vesicle dries. Some of the pocks, but never a large number, result in depressed cicatrices.

Fever

no.

The period of invasion is represented by febrile reaction, which is generally very slight, and within twenty-four hours the eruption shows itself—most commonly on the chest, but soon also on the face, trunk, and limbs. The spots are not very numerous, but fresh ones form for two or three days after their first appearance, and altogether they number, as a rule (according to Thomas), from 50 to 200. A few vesicles form on the mucous membrane of the mouth, palate, and lips. Whatever fever preceded the vesicles continues for a few hours, or for two or three days; it is generally not above 102°, but may reach 104°. The lymphatic glands of the neck may be enlarged. Death very rarely occurs, but convalescence is not always rapid.

Gangrenosa

In *varicella gangrenosa* some of the vesicles increase in size, become purulent, form reddish brown or black scabs under which the skin sloughs, and ultimately leave circular ulcers with clean-

Complications.—As a result of mumps, it happens occasionally that the testicles become inflamed just as the parotitis is subsiding, but it may be earlier or later than this. It is more common in adults than in boys. The process begins in the epididymis, the testicle swells, and there may be effusion into the tunica vaginalis, and oedema of the scrotum; it is accompanied by a rise of temperature, sometimes considerable. The inflammation subsides in a few days, but it may be followed by a permanent atrophy. More rarely there is double orchitis.

There seems to be some doubt whether a corresponding affection of the mammae or ovaries occurs in females.

The **Anatomical Change** in mumps is an inflammatory infiltration, serous and cellular, of the inter-alveolar fibrous tissue of the salivary glands.

The **Diagnosis** presents no difficulties, and the **Prognosis** is favorable.

Treatment.—The patient should be confined to one room, or even to bed, and food must be in a fluid form. Locally, fomentations generally give relief, and an antiseptic mouth-wash, containing boric acid, should be used frequently. Internally, small doses of potassium citrate or ammonium acetate may be grateful to the patient.

WHOOPING-COUGH.

(*Pertussis.*)

Whooping-cough is a disease characterised by a peculiar convulsive cough, followed by a long-drawn inspiration through the nearly closed glottis, by which a crowing noise, or "whoop," is produced.

It is contagious, generally requiring rather intimate contact, but sometimes apparently conveyed by clothing, and sometimes certainly by the sputum. Children are very susceptible, and most people have the disease in early life, while it quite rarely attacks adults. It is protective against a repetition of the disease, even more so, perhaps, than the exanthems. It is most common between the ages of one and eight years, and girls are more liable to it than boys. It occurs in epidemics, but there is not much evidence that such epidemics are determined by climate or weather. It has often been observed that an epidemic of whooping-cough has immediately followed an epidemic of measles.

Symptoms.—The period of *incubation* is about ten days. The first stage is one of bronchial catarrh, which is not always distinguishable from an ordinary catarrh induced by exposure to cold. There are cough, expectoration in children old enough, a few rhonchi in the chest, and slight pyrexia; but sometimes with the cough there is an unusual repetition of the expiratory effort, which may lead to suspicion. This preliminary bronchitis lasts from seven to ten days, and then there is a more or less rapid transition into the whooping stage. F[illegible] drawn inspiration follows the cough, and [illegible] "whoop." But the cough itself is as characteristic [illegible] The child may be playing with its toys, apparently [illegible] suddenly stops, seems distressed for a moment [illegible] runs to its mother or nurse. A short cough [illegible] followed by another and another without any [illegible] tion, each successive cough getting less [illegible] until they have mounted up to fifteen or twenty [illegible] in the course of seven or ten seconds. Then [illegible] inspiration with loud laryngeal sound, the [illegible] of short coughs succeeds, with another "whoop," [illegible] may occur once or twice more, with less violence [illegible] finally a little tough mucus is expectorated. [illegible] During the coughing efforts, the face becomes [illegible] the features swollen, the eyes starting from [illegible] hanging from the mouth, blood-stained [illegible] directions, and little relief takes place even [illegible] until the final expectoration of mucus [illegible] paroxysm. During this time the child [illegible] absolutely uncontrollable reflex process [illegible] they feel the attack coming on, will seize [illegible] it under their mouths, and in another [illegible] entirely at the mercy of the cough, and [illegible] on about them. As a result of the [illegible] during the coughing efforts, hæmorrhages [illegible] bleeding from the nose, mouth, or [illegible] mosis, petechiæ under the skin, and [illegible] hæmorrhage. After a time the face often [illegible] bloated appearance from the frequent [illegible] of blood to the chest. Sometimes a small [illegible] frænum linguæ, from the pressure of the [illegible] during the cough. The attacks often appear [illegible] but they constantly occur if the child cries or [illegible] or even if the child is disturbed, as when the nurse [illegible] off the clothes for an examination of the chest. [illegible] occur both day and night, and it has generally been [illegible] they are more frequent in the night hours, between [illegible] 6 A.M., than during the other twelve hours of the [illegible] tions in the whooping-cough ward of the Evelina [illegible] sick children did not confirm this. The number [illegible] which may, as above shown, include three or four [illegible] ranges from one to sixty in the twenty-four hours [illegible] rare to have forty attacks, and many cases never reach thirty in the twenty-four hours. In the intervals the child may be perfectly well, and is free from fever, unless there is some complication; the appetite also may be good, and the child soon replaces what he loses by vomiting, which does not, as a rule, occur in more than a small proportion of the paroxysms. This second stage of whooping-cough lasts a variable time, often from three to six weeks, but it may be three months or even more. The attacks gradually get less frequent, until they cease altogether, or as they diminish they may be accompanied with attacks of simple cough, not followed by a whoop, and this may last a few weeks longer. Death rarely occurs directly from the paroxysms; it may do so from prolonged closure of the glottis, or from cerebral hæmorrhage. Other **Complications and Sequelæ**, however, occur, which make whooping-cough a serious and even dangerous complaint. Amongst the former may be classed bronchitis, which may continue throughout, and broncho-pneumonia, which is revealed by high fever of remittent type, by crepitant râles or patches of dulness or tubular breathing, and by continued dyspnœa in the intervals between the cough. Often, but not always, the whoop is absent during broncho-pneumonia, as it is if any other febrile complication ensues. General convulsions sometimes occur either as a direct result of the whooping attack; or less commonly as the indication of cerebral hæmorrhage or thrombosis; or, it may be, of the onset of pneumonia. As sequelæ, continued bronchitis, emphysema, and tuberculosis of the lungs occasionally occur.

Pathology.—The nature of the disease is still obscure. It has been regarded as a purely nervous affection, and as due to pressure on the vagus by swollen tracheal or bronchial glands; but it obviously has very close similarities with the other zymotic diseases. It is, indeed, highly probable that it is due to a micro-organism, but the elliptical cocci found by Bürger in the sputum, and the bacillus described by Afanasieff in the respiratory mucous membrane have not been proved to be causative of the disease. If irritation of nerve-terminations will explain the cough it is not so easy to find a cause for the closure of the glottis, which produces the whoop. It is generally thought to be spasmodic, but Goodhart suggests that it is only a passive approximation of the cords, or a failure to open freely when the sudden inspiration takes place.

The **Morbid Anatomy** of pertussis is that of its complications, chiefly broncho-pneumonia. The laryngeal and tracheal mucous membranes are injected, and the bronchial glands are swollen.

Diagnosis.—This mainly depends on the whoop, on the convulsive character of the cough, and on the regularity of the course from the catarrhal to the convulsive stage. Enlarged

Incubation [illegible]

Glands

period of from five to seven days. The patient is taken suddenly with stiffness in the neck, difficulty of swallowing, and febrile reaction, with anorexia and perhaps vomiting. The fauces are little, if at all, affected, but on the second or third day of pain the anterior cervical glands along the border of the sterno-mastoid muscle of the left side are found to be enlarged and tender. In another day or two those of the right side are swollen, and the posterior cervical, axillary, and inguinal glands may be also involved. There is generally abdominal pain, and tenderness; and the liver, spleen, and mesenteric glands are enlarged. The glands begin to get smaller after from two to five days. The temperature may reach 104° on the third day, and it will continue high as long as the glands remain enlarged. Constipation is often troublesome. The disease subsides usually without complications; but nephritis sometimes occurs, and convalescence may be retarded by anæmia. The bacteriology of the disease is at present negative.

The treatment consists in rest, a simple diet, the relief of the constipation by small doses of mercury or salines, and the use of preparations of iron during convalescence.

INFLUENZA.

This term, often wrongly applied to any severe nasal catarrh, is the name given to an epidemic disease which in past times has frequently swept over Europe, but after the violent epidemic of 1847–48 was practically unknown among us until the winter of 1889–90, when a similar outbreak occurred. On this occasion the disease appears to have been first observed in Bokhara in the preceding May; it appeared at St. Petersburg in October, and soon invaded Austria, Germany, France, England, and other European countries, as well as the United States of America. A few months later it was conveyed to India, Australia, New Zealand, the African Coast, and South America. The disease has again broken out in successive years and latterly has rarely been entirely absent.

Rapidity

Ætiology.—The true epidemic invasions of influenza have always been characterised by the extraordinary rapidity with which the population has been attacked, especially in crowded towns. Hundreds have been struck down at the same time, or within a few days, and this, among other circumstances, led to the view that the disease was not contagious from man to man, but was borne by the air simultaneously to many people. This feature was especially marked in the first of the recent epidemics. But there is no doubt that the spread of the disease is determined by peripheral neuritis, parotitis, meningitis, tremors, weakness, paralytic affections, mental derangements, and others.

Duration
Relapse

From the disease in its simplest or uncomplicated form, many patients recover by the third or fourth day, or remain weak for a few days longer; but there is sometimes a relapse about the end of the first week.

Bug

Pathology.—As influenza is rarely fatal except through one of its inflammatory complications, its morbid anatomy is generally comprised in that of the lesion, such as pneumonia, which has immediately caused death. There is little doubt that the disease depends upon a specific microbe. In addition to the streptococci, staphylococci, and pneumonic diplococci, which are not distinctive, a minute bacillus has been discovered by Pfeiffer in the sputum of influenza patients, and this has been successfully cultivated by Kitasato.

Pneumonia
typhoid

Diagnosis.—The great variety that influenza presents will lead to its being diagnosed in the early days of an illness, when further acquaintance with the case may show it to be some other febrile complaint, such as pneumonia, and especially enteric fever (*see* p. 47). (The very sudden onset, the local pains, and the short fever are the chief distinguishing points of influenza.)

Bed
pains

Treatment.—The patient should save his strength by at once taking to his bed. In the early stages the severe pains call for treatment, and may be met by sodium salicylate (10 to 15 grains every four or six hours), or by antipyrin (5 or 7 grains). The great tendency to prostration after the illness makes it necessary to give these drugs with caution. Instead of them salines (10 or 15 grains of potassium citrate, or half an ounce of liquor ammonii acetatis) may be given in the early stages, combined with expectorants, if there is much bronchial complication (ammonium carbonate, 3 to 5 grains, or tincture of senega, ½ to 1 drachm). As the fever subsides, most cases require a tonic regimen. Quinine and nux vomica are especially useful; and in older patients stimulants are also needed.

Cessant
Tonic,
Stimulants

Local

The local manifestations of the disease require to be treated as they would be if arising under other circumstances.

CEREBRO-SPINAL FEVER.

(*Epidemic Cerebro-Spinal Meningitis.*)

Place

This disease has been known only since the beginning of the present century, when it occurred in Geneva. Of late years it has been common in Germany. In 1846 it appeared in Ireland,

and again in a severe form in 1866–1868; but only isolated epidemics have occurred in England.

Ætiology.—Very little is known of this. The disease is not apparently contagious from man to man directly, and it has been suggested that the virus may be transmitted through one of the lower animals. The disease attacks young people with no great distinction of sex. In epidemic times, the presence of an acute disease, especially pneumonia, may act as a predisposing cause.

Symptoms and Course.—There are in a few cases slight premonitory symptoms, such as headache, nausea, or malaise; but mostly the disease begins suddenly with severe headache, and sometimes a rigor, so that the patient has to give up at once, and suffers also from pain in the back and limbs, vomiting, and fever. The headache is chiefly occipital, but may be frontal or temporal; with it is stiffness of the muscles of the back of the neck, and, indeed, the head is drawn back by the contraction of the deep muscles; and the dorsal and lumbar muscles may be similarly affected, so that the back is kept straight (*orthotonus*), or even arched with the concavity backwards (*opisthotonus*). Pains frequently extend down into the muscles of the lower extremities, and cutaneous hyperæsthesia may be also present. In addition to these symptoms, referable to irritation of the roots of the spinal nerves, there are others due to the implication of the cranial nerves. These may be, in different cases, ptosis, or strabismus; contraction or dilatation, or inequality of the pupils; or contraction of the facial muscles; but trismus is rare. Optic neuritis and purulent irido-choroiditis occur, and conjunctivitis and keratitis, probably from external irritation. Pain in the ear, tinnitus and defective hearing are not uncommon, and suppuration of the labyrinth or of the tympanum may occur. Deficiency of the sense of smell has been noted. Drowsiness, delirium, and coma, with or without convulsions, supervene in due course; and death takes place with varying rapidity in different cases. Fever is present from the first, but it runs no regular course; it is remittent or intermittent, perhaps normal for a day or two, and then rising to 102° or 103°, seldom exceeding 104°. Occasionally the fatal termination is preceded by a temperature of 108° or 109°. With recovery the temperature falls slowly and irregularly. An important feature of epidemic meningitis is the occurrence of cutaneous eruptions, of which herpes facialis is the most frequent. It appears early in the illness, as frequently in severe as in mild cases, and in more than half altogether. It often covers a large part of the face, and may be bilateral; and sometimes it affects the trunk or limbs. Urticaria, erythema, and purpura also often occur. All these eruptions may appear at the same time, and they are often symmetrically arranged. Sometimes the joints are inflamed, hot, red, painful, and swollen—a condition which generally subsides. The abdomen is often retracted; the spleen is not often enlarged. There is not infrequently polyuria, and the urine may contain a little albumen, or a trace of sugar. The pulse is generally quick, and perhaps irregular.

Joints
Abdomen
urine
Pulse

Varieties.—Some cases are fatal in a few hours or days (*foudroyant*, fulminant or explosive); others, on the contrary, are abortive, getting well rapidly in a few days; the majority last from two to four weeks. Remittent and intermittent forms are recognised, in which the fever is much less or absent for periods of two or three days at a time; and there is a "typhoid" form with muttering delirium, dry brown tongue, involuntary evacuations, and bed-sores. In the distribution of the other symptoms considerable variety occurs. The mortality varies from 30 to 70 per cent. in different epidemics.

Fulminant
[illegible]
Majority
Remittent
Intermittent
Typhoid

Sequelæ.—Deafness is most common from the lesion of the internal and middle ear, and if both ears are destroyed in very young children deaf-mutism necessarily results. Vision is also often impaired. Chronic hydrocephalus is another result of the preceding inflammation, and may have for its symptoms headache, convulsions, mental deficiency, and weakness of the limbs. Hemiplegia, paraplegia, and aphasia also occur, but are generally of a transitory nature, if first appearing within a short time of the illness.

Morbid Anatomy.—There is an acute leptomeningitis of the brain and spinal cord. The pus and lymph are most abundant on the convexity of the brain, along the larger blood-vessels and in the fissures. In the spinal cord the posterior surface is more affected than the anterior, and the lumbar region more than the other parts. The ventricles of the brain contain turbid serum or pus. Punctiform hæmorrhages, accumulations of leucocytes, or actual abscesses are found in the cortex of the brain. Other changes found are congestion of the lungs, liver, spleen, and kidneys, fatty degeneration of the renal epithelium, and granular degeneration of the voluntary muscular fibres; sometimes ecchymosis of the pericardium and pleura, and suppuration of the joints. The diplococcus pneumoniæ, streptococci, and the meningococcus intracellularis have been found in the pus from the meninges.

Diagnosis.—This is not difficult in the middle of an epidemic. The characteristic features are the sudden onset, the headache, pain in the back and limbs, stiff neck, and the herpes labialis. The purpuric eruption also seems to distinguish it from other forms of meningitis, tubercular and suppurative, which must always be carefully considered. The fact that it occasionally complicates pneumonia must be remembered, but must not have too much weight in quite young children, in whom retracted head and convulsions may occur from pneumonia alone.

Treatment.—This must be conducted on the same principles as that of other forms of meningitis, to which the reader is referred.

AGUE AND MALARIAL FEVERS.

Especially in marshy districts, but also in other localities possessing special features, certain diseases occur endemically, which are known from their clinical characters as *intermittent* or *remittent* fevers, and from their source as *malarial* fevers. The word *malaria*, meaning primarily the bad air (mala aria) which is exhaled from the surface in certain districts, has been also used to indicate the virus or infective agent, till recently unknown, which is contained in such air, and which, by gaining access to the individual, gives rise to one or other of the fevers in question.

The best known of these is *ague* or *intermittent fever*, characterised by the periodic recurrence of "fits," or febrile attacks, separated by intervals of comparative or almost perfect health. The attacks last a few hours, and recur daily or every other day, or every third day; and the subsidence of the temperature to normal after each attack constitutes the intermission from which its name is taken. But no broad line can be drawn between the two classes of malarial fevers: that in which the temperature falls to normal between the attacks (intermittent), and that in which the temperature only falls partially (remittent).

Ætiology.—The prominent fact in their ætiology is their frequent association with marshy lands (*paludal* or *marsh* fevers); hence their prevalence in certain countries and districts. For instance, in England, the borders of the Thames in Kent and Essex, and the fens of Cambridgeshire and Lincolnshire; on the Continent, the flat district of Holland and North Germany, and the west coast of Italy; in Africa, the west coast; in Asia, many portions of India, especially along the Ganges and the Indus, and also parts of China and Persia. These present for the most part large areas of low-lying land, with more or less abundant vegetation, partially or entirely submerged. But it is not so much the mere presence of water that determines the various forms of ague as the moistening of the soil and vegetation with water, and its subsequent exposure to the heat of the sun—alternating processes, which lead to decomposition, and thus probably favour the development of the organism to which these diseases are due.

A high temperature, therefore, has considerable influence, and, as a fact, malarial diseases are confined within certain latitudes—namely, 63° north latitude and 57° south latitude—while the most severe forms of all occur in the tropics. The seasons of the year at which malarial fevers are most prevalent, also, are chiefly the hotter seasons; but in different countries the occurrence of rains at particular times may modify the result. As already stated, these fevers occur in other than low marshy districts. Thus they have been observed in the Apennines at a height of 1100 feet above sea level; in the Pyrenees, at 5000 feet; and in Peru, at 10,000 feet. The circumstances are then found to be essentially the same as in the case of marshes—that is, a loose porous soil, resting on a clay bottom or rocky base, so that the water is retained, and all the conditions exist necessary for the alternate moistening and drying of the soil. Marshes impregnated with salt water seem to be more noxious than those saturated with fresh water.

The infective agent is contained in the emanations from such soils, and is probably, as a rule, conveyed into the body through the process of respiration; though there is evidence that ague can be introduced by drinking the marsh water itself. The disease is active at all periods of life, and no race appears to be absolutely insusceptible, though negroes are less liable than white men. Those more susceptible to its influence are people in ill health, those exposed to damp, to cooling influences, or to excessive heat of the sun, and those who indulge in immoderate eating or drinking.

One attack predisposes to another, and those who have suffered in ague districts may, long after removal, be again attacked on the occurrence of slight causes. Possibly the poison lies latent in the system, as it has been observed that a person may be a short time in an ague district, leave it without having an attack, and afterwards, in a perfectly healthy locality, develop the disease.

Symptoms.—The period of *incubation* is from three to twelve days, shorter in the irregular, longer in the regular forms; but it may break out almost immediately on exposure. In some cases there are *prodromata*, consisting of malaise, headache, pains in the limbs, epigastric fulness, nausea, and slight chills or flushes.

The ague fit has long been described as consisting of three stages: the *cold* stage, the *hot* stage, and the *sweating* stage.

The Cold Stage.—The patient feels tired and listless: has headache and pain in the back and loins, then feels chilly, and the *rigor* begins. He generally lies curled up in bed, shivering all over, and his teeth chattering. The face is blue and pinched, the nose and ends of the fingers livid, and the skin in a condition of *cutis anserina*. The pulse is small, hard, frequent, and irregular; the breathing quick and shallow. The surface is actually cold, but the thermometer, placed in the mouth, rectum, or axilla, will show that the temperature is already considerably above normal. Indeed, it commences to rise some minutes (or even one or two hours) before the rigor; but the sensation of cold and the actual cold of the surface are due to contraction of the superficial vessels. The urine is abundant, clear, and of low density. This stage lasts from half an hour to two hours, and the axillary temperature rises rapidly, attaining a height of 105°, 106°, or even more, towards the end of the period.

The Hot Stage begins with a sense of warmth diffusing itself over the body, and the surface, hitherto cold, becomes intensely hot. The temperature in the axilla rises still a little higher than it was at the end of the cold stage: the arteries are relaxed, the pulse becomes quick, full, and hard; the carotids throb, the face is flushed, and the head aches. There is a tendency in some cases to stupor or delirium. The urine during this stage is scanty, dark, and of high density. There is often an eruption of herpes about the mouth. This stage lasts from three to four hours.

The Sweating Stage.—The skin, hitherto dry, now gradually becomes moist. Sweating begins first on the face, and then spreads to the rest of the body, and continues profusely from one to two or three hours, the pains and discomfort of the hot stage are relieved, the pulse becomes softer and slower, and the tongue moist. The temperature falls at first slowly, then more rapidly, until the normal is reached; and, finally, with the subsidence of the temperature the sweating ceases, and there is a return to the preceding state of health. During this stage the urine is of high density, and deposits a sediment of urates.

In ordinary cases the spleen enlarges during the cold and hot stages, so that its area of dulness is increased, and it may be felt below the costal margin. The attacks are not always thus complete. There may be only slight chill, but a hot stage of two or three hours, with no sweating; or there may be chill, with sweating, but no hot stage; or cold and hot stages may be absent, and sweating with a slight rise of temperature may alone represent the attack. When the attack is over the patient feels perfectly well; but after a variable interval he is seized with another similar paroxysm.

Forms of Ague.—In some cases the attacks occur every day—this is called *quotidian* ague. In others the attacks occur every other day, with an intervening day of no fever; in others, again, at intervals of three days, with two days of health intervening. The former are called *tertian* agues (the second attack occurring on the *third* day), the latter are called *quartan* agues. There also occur more rarely cases described as *double quotidian*, in which two attacks occur daily; *double tertian*, in which the attacks are daily, but the fevers of the first, third, and fifth days resemble each other, and differ from those of the second, fourth, and sixth days; *double quartan*, in which attacks occur on the first, second, fourth, and fifth days, and so on; and *semi-tertian*, in which a quotidian and tertian ague appear to be combined, two attacks occurring on the first day, one only on the second day, two on the third day, and one on the fourth.

Tertian ague is more common in temperate climates; quartan is less frequent than the others, and it is said not to occur in the tropics. The attacks take place mostly in the morning or noon hours, the tertian especially at noon; succeeding attacks are, however, not always at the same hour, but in some cases may get earlier and earlier—in others, later and later. The former are said to *anticipate*, the latter to *postpone;* and the character of the fever may be thus changed, the tertian becoming quotidian, or the quartan tertian.

Some other symptoms that occur in association with ague are regarded by writers as manifestations of malarial poisoning, and the cases have been called *masked intermittent*. Such are especially different forms of *neuralgia*, of which attacks may occur on alternate days, or themselves alternate with definite paroxysms of ague. The supra-orbital division of the trigeminal nerve is the one most often affected, the second and third divisions less frequently. Sciatica, thoracic neuralgia, and cardialgia also occur. It is said that attacks of the following, among others, may represent masked agues:—sneezing, coughing, hiccough, tremors, convulsions, temporary paralysis, aphasia, delirium, hallucinations, maniacal attacks, and sleeplessness; or bronchial catarrh, skin eruptions, and hæmorrhage from the mucous membranes. But some writers throw doubt on the nature of the association between them.

Ordinary attacks of tertian ague are not generally fatal, though death may happen in very young or in old people, or in those debilitated by previous illnesses. If the spleen is very much distended it may rupture spontaneously or after injury, and the blood will escape into the peritoneal cavity; or hæmorrhage may occur in its substance, followed by suppuration and rupture of the abscess into the peritoneum. But other dangerous forms occur, and these are generally distinguished by the special implication of certain organs, such as the nervous system, the bowels, or the lungs. In these so-called *pernicious* forms, the more serious symptoms are not generally developed at the beginning of an illness, but only after the lapse of some days. Among forms involving the nervous system may be mentioned a *comatose* form: there are marked headache, dizziness, apathy, or even drowsiness in the cold stage; and these pass into complete unconsciousness in the hot stage, with rapid, stertorous breathing, wide, immovable pupils, and limbs completely relaxed. The patient may lie thus for ten or twelve hours, or more, and then gradually recover consciousness in the sweating stage. In other cases there may be

The Parasites of Ague.—Malarial fevers are undoubtedly due to the action of a micro-organism first described by Laveran—viz., the *Hæmatozoon*, or *Plasmodium malariæ*. The organisms known by this name are protoplasmic bodies which exhibit amœboid movements, and are first seen in the early part of the intermission within (or adherent to, Laveran) the red corpuscles, looking like clear spaces. In the course of a few hours they enlarge, and contain a few pigment granules, while the body of the red corpuscle becomes paler, its hæmoglobin having been extracted and changed into black pigment. A little later, and still during the intermission, the plasmodium enlarges, so as to obscure the decolorised corpuscle, and itself contains more pigment.

Then the pigment aggregates towards the centre of the plasmodium, and a process of division, or *segmentation*, takes place, so that it breaks up into from six to fifteen or twenty smaller clear bodies; and, at the same time, both these and the pigment granules become free in the blood plasma. This process of segmentation corresponds with the onset of the paroxysm of ague; and during its course the blood becomes free of the organisms, which are believed to reach some solid organ, such as the spleen, whence they subsequently emerge to attack fresh corpuscles, and to start another fit of ague. Other bodies have also been found in the blood of sufferers from ague—viz., *flagellated* bodies and *crescentic* bodies. The former are spherical in shape and provided with filaments which undergo lashing movements; and which may also be seen free in the plasma. The latter are colourless, transparent, immobile, with a few pigment granules in the interior; they are generally longer than the diameter of a red corpuscle, but some smaller ones may be seen inside corpuscles. The above facts explain the destruction of the corpuscles, the anæmia, which is so characteristic of the malarial fevers, and the occurrence of pigment in the blood, to be mentioned presently.

In addition to this it has been found that the organisms present some differences in different forms of malaria. The plasmodium of quartan ague has slower amœboid movements, develops more slowly in the corpuscle, taking about three days to fill the corpuscle against two on the part of the tertian parasite, forms larger pigment granules, and breaks up into a small number of segments (six to twelve), whereas the tertian parasite forms twelve to twenty segments. Quotidian ague is sometimes due to alternate outbreaks of the tertian parasite, at others to repeated crops of quartan parasite. In the irregular forms, including so-called *æstivo-autumnal* fevers, the parasite in later stages looks more like a ring, has less pigment, and remains of smaller size: crescents and flagellated bodies are numerous.

The organisms may be with care detected in a drop of the fresh blood, obtained by puncture of the finger (or in special cases from the spleen), placed between cover-glasses, and magnified 500 diameters. Staining with eosin and methylene blue is also effectual.

Malarial Cachexia.—In those who have had repeated attacks of the disease, and in some who have resided in malarial districts without developing such attacks, the bodily health is seriously affected. The most prominent feature is *anæmia*, causing a sallow earthy look, with pallor of the lips. With this are numerous functional disturbances, dizziness, loss of appetite, digestive disorders, pains in the joints and muscles, lassitude, and indisposition for exertion. In severer forms there is ascites, œdema, or hæmorrhage; but no fever. The spleen in these cases is enlarged, reaching even below the level of the umbilicus, and forward to the middle line, constituting the *ague-cake*. It is hard and often tender. The liver may be also enlarged.

Morbid Anatomy.—As death rarely takes place in simple cases of intermittent fever, the pathological anatomy of this disease is chiefly known from the severer and more complicated cases. In the former the spleen is simply enlarged. In old cases and in malarial cachexia this organ has become more fibrous, firm, tough, and pigmented; often with a thickened capsule, adherent to surrounding parts, presenting infarcts, and in long-standing cachexia perhaps lardaceous. The liver may be enlarged, especially in cases with jaundice.

In severe or prolonged cases the blood may contain brown or black pigment granules, either free or within the white blood-cells; and these granules are found not only in the blood but also in the solid organs, such as the spleen, liver, kidneys, brain, heart, and in the lymphatic glands, and marrow of the bones, giving a slaty or dark grey colour to the various tissues. The condition is described as *melanæmia*. The masses of pigment may be large enough to block up the capillaries.

The **Diagnosis** of ague is generally easy: the attacks are distinctive in themselves, and their nature is confirmed by the recurrence after a healthy interval. *Pyæmia* may resemble it in the rigors and sweating with rise of temperature, and in the subsequent remission and even intermission; but the attacks are generally less regular, and long intervals of apyrexia do not commonly occur in pyæmia. The pyæmic attack, also, is said to begin more gradually and to subside quickly. Pyrexial attacks from suppuration and the hectic of phthisis may simulate ague. On the other hand, in the severe forms of ague the malarial origin may be overlooked, from the prominence of some local disorder. Thus the comatose form may be taken for apoplexy, and other forms may be mistaken for pneumonia or cholera.

Treatment.—Quinine is the remedy which, in the large majority of cases, effectually controls ague, and is also used to prevent its occurrence in those exposed to the malarial poison. For the latter purpose the sulphate of quinine may be given in doses of 2 to 5 grains three times daily.

If there are indications of the onset of the disease, 10 grains may be given every night for three or four nights, and will sometimes prevent the outbreak. In the developed disease, 20 to 30 grains should be given in the day, and it is generally thought better to give this in two or three large doses rather than in small quantities at frequent intervals. The last big dose should be given about six hours before the next expected attack. In the severe or pernicious cases still larger quantities of quinine may be necessary, and no prominence of the local symptom should lead one to abstain from its use. If quinine is vomited it may be given with a little opium, or may be injected per rectum. It has also been given subcutaneously in doses of 2 to 3 grains for an ordinary attack. The solution may contain 1 grain to 2, 3, or 5 minims of water with a little dilute hydrochloric acid; but it often causes irritation of the skin, with the formation of abscess.

In a certain number of cases quinine does not completely cure, and arsenic in doses of from 8 to 12 minims of the liquor two or three times daily has been found useful. An alcoholic extract of eucalyptus, the sulphate of bebeeria, and piperine have also been used, but with doubtful efficacy. During the attack little can be done but minister to the comfort of the patient and relieve symptoms. In the cold stage the patient must be kept warm by sufficient clothing and hot bottles to the feet; and he may be ordered some diffusible stimulant if there is a tendency to collapse. In the hot stage the coverings will have to be removed, the body may be sponged with cold or tepid water, and thirst satisfied with cooling drinks.

Malarial cachexia requires removal from the infected district, and internally the use of iron and arsenic; Maclean recommends the baths of Carlsbad and Homburg. Enlarged spleen is said to be amenable to the cold douche directed upon it, or to the ointment of red iodide of mercury smeared over the surface, which is then exposed to the fire till it begins to smart. The continuous current of galvanism may also be applied. Internally quinine, iron, and iodide of potassium should be given, or the phosphates of iron, quinine, and strychnia.

To treat [illegible] – [illegible] [illegible] [illegible]

DENGUE.

Dengue, or Dandy Fever, is a disease occurring only in or near the tropics. It begins with fever of short duration, accompanied by pains in the joints and limbs; then, after a short interval of apyrexia, there is a second fever, and often a cutaneous rash. Second or third relapses even occur.

m (from one to three days) probably explains, as in , its rapid spread in unprotected areas. The patient is seized with pain in one or other joint, often in the finger, dache and fever. Other joints are afterwards affected, pains shift about, lessening in one part as they appear er, and affecting the muscles as well as the joints. The is accompanied by pains in the eyeballs. The tempera- s to 102°, 103°, or even 105°, and the pulse is commonly over 100. In many cases, also, this first fever is accom- y rash, either redness of the face, or a general red colour, throat may be sore. But the rash disappears in twenty- rs, and about the same time the pyrexia terminates— s suddenly with critical symptoms, such as sweating. s abate, and the patient is in comparative comfort, but two, three, or four days, when again he becomes fever- rash appears, which is either diffused like scarlatina, or d like measles, or elevated like lichen tropicus, or it may semble urticaria. It generally causes some itching. It the palms of the hands, and spreads to the whole of the t lasts from a few hours to two or three days, and is by desquamation. In this second fever, also, joint pains cur, and may persist or relapse after the subsidence of . The whole duration of the disease is about eight days, here second relapses follow, or where the joints remain painful, and deformed, as they may do for months after- ven becoming partially ankylosed. It is, as a rule, only infants and old people. Mild forms may present only sore throat, and the second or *terminal* eruption. In forms there may be coma, hyperpyrexia, failure of the lema of the lung, or cyanosis.

is known of the pathology of the disease. From the l joint pains it has been called *scarlatina rheumatica*.

of the second rash by the use of camphorated oil. During convalescence iron and quinine should be given.

BERI-BERI.

(*Kakke.*)

This is an endemic disease, consisting essentially of a multiple or peripheral neuritis, and, in all probability, due to a micro-organism.

Ætiology.—The important feature in its causation is its relation to locality. It has been observed chiefly in Japan, China, the Malay Peninsula, and slightly in India and Ceylon; in some parts of the east coast of Africa, in the West India Islands, and the northern parts of South America. The most susceptible time of life is between 20 and 30, and males are more liable than females. The disease occurs in outbreaks, which may be determined by importation of an individual from elsewhere; but it does not seem to be transmitted directly from man to man, nor to be conveyed in food or water. It seems, like ague, to arise from the soil; but, unlike ague, it is frequent in towns and inhabited places, and it is promoted by overcrowding.

Symptoms.—These are, for the most part, the weakness or paralysis of muscles, muscular atrophy, and sensory disturbances, which are characteristic of multiple neuritis (*see* p. 179); but there are added in this form certain features not commonly seen in the familiar cases of neuritis due to alcohol, diphtheria, or plumbism—namely œdema of the legs, or even extensive anasarca, and severe or fatal dyspnœa from cardiac failure or œdema of the lungs.

There is, however, considerable variety in the effects produced by these lesions: in some anasarca is pronounced or extreme (*wet beri-beri*); in others, the muscular atrophy is the chief feature, and the patients are thin and emaciated (*dry beri-beri*); and intermediate conditions occur.

The disease often begins slowly with a state of languor, with weakness of the legs and knees, pains in the calves, and slight dyspnœa; or the occurrence of weakness and numbness of the legs, and pains in the calves may be almost sudden. As the disease develops, there are loss of power and muscular atrophy beginning in the extensors on the front of the leg, and then affecting the other muscles of the leg and thigh, as well as later, the extensors of the hand, the biceps, and, it may be, the abdominal muscles, the diaphragm, and the intercostals. The knee-jerk is generally soon lost. When the legs are chiefly affected, the patient has a gait characteristic of foot-drop: the heel is lifted high to clear the ground, and the toes come down before the heel. Anæsthesia is early noticed in the skin over the tibiæ, and may extend to other parts of the limbs and trunk. Hyperæsthesia of the muscles, especially of the calves, tenderness of nerve-trunks, and painful cramps occur, as they do so often in multiple neuritis from other causes. A little œdema over the shins appears to be almost invariable. Sometimes it extends to other parts, or to the whole body. When anasarca is extreme the urine is scanty. There is in most cases palpitation and dyspnœa, and bruits are heard at one or more of the cardiac orifices. The pulse is soft and rapid. The temperature is normal, or even subnormal.

Cases often last about five weeks; some a shorter time, others as much as twelve months or more. The danger lies in cardiac failure, with rapid irregular action, or in asphyxia from œdema of the lungs: and these conditions sometimes arise quite suddenly and carry off the patient. Sometimes fatal exhaustion follows vomiting.

Morbid Anatomy.—*Post-mortem*, in addition to the œdema and anasarca seen during life there are ecchymoses under the serous membranes, in the muscles, and in the sheaths of the nerves; the lungs are engorged and œdematous, the right side of the heart dilated, and the muscular fibres possibly degenerated. The muscles are either atrophied or swollen, and the nerves show evidence of inflammation or neuritis.

Treatment.—There is no specific for this disease. The essential thing is the removal to an uninfected locality; or, failing that, to a well-ventilated apartment high above the soil. Little more can be done in the early stages; in the later, iron and strychnine internally, and faradisation and massage to the limbs are recommended. For serious cardiac failure active purgatives, full doses of nitro-glycerine or nitrite of amyl, and, if necessary, bleeding should be employed.

YELLOW FEVER.

Yellow fever is an acute specific disease, occurring within certain geographical limits, and characterised by fever of short duration, a yellow tint of skin, severe gastro-intestinal disturbance, and albuminuria, or suppression of urine.

Ætiology.—This disease was first met with in 1647 in the West Indies, and is peculiar to that group of islands, to North and South America, and to the west coast of Africa. It has, indeed, been occasionally imported to other parts of the world—for instance, in 1865 to Swansea, where a slight epidemic was the result; but it has never maintained itself away from the localities mentioned, which lie between the latitudes of 48° north and 35° south. It occurs almost exclusively in crowded towns, and especially in those having a maritime commerce; and for the most part it is confined to low levels, and is rarely found higher than 2000 feet above sea level; but epidemics have occurred in the Andes at an elevation of 11,000 and 14,000 feet.

It requires a high temperature, not less than 70° F. or 72° F., according to most writers, but it has been known to occur at lower temperatures, *e.g.*, 65°. It is stated to be stopped absolutely by cold sufficient to freeze the earth. It is most fatal in the summer months—from May to August.

It is still disputed whether yellow fever is contagious from man to man, or is conveyed by the excreta in the same way as typhoid fever and cholera. Certain it is that one sick person arriving in a new locality may be the source of an outbreak among the former residents, and that the virus in some form adheres with great tenacity to clothes, and to other structures, such as the hull and other parts of ships. It affects all ages and both sexes; but negroes are less susceptible to it than whites. It rarely attacks the same individual a second time.

Symptoms and Course.—Its period of *incubation* is variable. According to Sternberg, it does not usually exceed four or five days; some authors believe it may be very much longer.

Sometimes suddenly, sometimes after premonitory symptoms, such as languor, headache, and malaise, there are chills or rigors of more or less severity. These are followed by febrile reaction, the temperature rising in two or three days to 105°, or even higher. There are generally frontal headache and severe lumbar pains, or pains in the joints. The pulse varies from 100 to 120, mostly not so quick in proportion to the temperature as in some other fevers. The tongue is generally covered with a thick creamy fur, leaving the edges and tip bright red. There are mostly tenderness and pain in the epigastrium, with nausea or vomiting. About the second or third day the conjunctivæ become yellow, and jaundice spreads to the whole body. The urine is scanty, and constantly contains albumen, which may be found as early as the second day of the illness. Bile-pigment appears a few days later.

On the fourth day there is often a remission of temperature, and the general pains subside. This may be the commencement of convalescence, the yellow tint gradually clearing up, albumen disappearing from the urine, and the patient recovering in two or three weeks. But in many cases the more serious symptoms continue.

The patient may fall into a fatal collapse; or the temperature may rise again for a few days, though not to the same height as before. The jaundice deepens, and petechiæ appear under the skin; the urine is still less in amount, while the albumen increases, and casts are present; and finally, complete suppression for days may occur. The vomiting becomes frequent. At first only the gastric contents, mixed with more or less bile, are discharged; but afterwards occur the so-called "black vomit," due to the presence of blood, often in a form which is likened to coffee-grounds. This is sometimes preceded by a limpid, ropy, opalescent fluid (white vomit). When blood is discharged by the stomach it is generally passed by the motions as well. Hæmorrhage may take place also from the nose, mouth, or gums; and the tongue by this time has lost its fur, and becomes dark brown, raw, and covered with blood crusts or sordes. Delirium becomes pronounced, or the patient may sink into coma. The mortality varies from 5 to 75 per cent. in different epidemics. Death takes place sometimes within a few hours of the onset, more often after the remission of fever, either from collapse, from profuse hæmorrhage, from coma, which is generally attributed to suppression of urine and uræmia, or from typhoid or adynamic conditions.

Anatomical Changes.—The changes described are anæmia and acute fatty degeneration of the liver; acute catarrh of the stomach, with ecchymosis, or hæmorrhagic erosions; hæmorrhages in the tissue of the lungs and under the pleuræ; pale, yellow-brown colour of the muscular substance of the heart, or acute fatty degeneration or ecchymoses; and acute parenchymatous inflammation of the kidneys, sometimes with miliary abscesses. The spleen differs strikingly from that of malarial disease, in being usually quite unaffected.

Sanarelli has isolated a bacillus (*B. icteroides*) from 2–4 μ in length, which appears to be the specific organism of yellow fever. He found it in the blood and tissues, but not in the alimentary canal. It is constantly associated with other micro-organisms (pyococci and *B. coli commune*), to which some of the secondary lesions of the disease are no doubt due.

Diagnosis.—There is generally little difficulty about the diagnosis in localities where the disease is prevalent. The early acute symptoms may present a certain resemblance to the onset of such illnesses as small-pox or pneumonia. Later, it has mainly to be distinguished from malarial fevers. These last are endemic, and not transmissible so as to affect healthy persons in new localities: they present intermissions of actual health, or, at least, remissions between the exacerbations, which recur with regularity; the spleen is enlarged, and the individual is not protected by his illness from future attacks.

Relapsing fever may be accompanied with jaundice, but the primary fever is longer, and the interval is one of very great improvement; the spleen is enlarged. Acute yellow atrophy begins more gradually, often with an apparently simple jaundice.

[illegible] a laxative or enema
make use of salines and diaphoreti
arise. High fever may be met by
internally, by very small doses of
lime-water; the action of the kidne
baths or vapour baths. The diet
and alcohol will be required; but wı
be given somewhat dilute, either as
as champagne. Sternberg recommen
great service in checking gastric irrita
of the urine, and giving favourable
coveries:—one-third of a grain of perc
grains of bicarbonate of sodium in 2 pi
8 tablespoonfuls are to be given every l

DYSENTERY

This is an acute inflammation of the
times of the lower part of the ileu
ulceration, and accompanied by fæcal
almost entirely of mucus, muco-pus, or b

Ætiology.—In the tropics, at least, it
an acute specific disease. It is enden
spreads in an epidemic form. It is wi
40° north and south of the equator tha
But it is not climate alone that deter
also the soil, since many places are exe
are favourable to it are
severer

It is probably contagious only in the same way as cholera and typhoid fever—that is, not directly from man to man, but only through the medium of the dejections, by which either the air or the drinking-water is contaminated. There is still much to learn about the cause of the disease. Some forms appear to be due to a protozoon, the *amœba coli*, which can be found in the stools, in the walls of the diseased intestine, in the associated hepatic and pulmonary abscesses, and in the sputa.

Symptoms.—In cases where it has been possible to fix the period of *incubation*, this has varied from a few days up to eleven. The patient is first taken with diarrhœa, and passes daily from two to five or six yellow or brownish-yellow loose fluid motions. There is some abdominal pain, and a certain amount of malaise, with loss of appetite. After three or four days the stools become suddenly more frequent, so as to reach ten, twenty, forty, or sixty or more in the twenty-four hours, and they acquire the special characters of *dysenteric stools*. Each discharge is very scanty, and may not be more than a few drachms; but the total quantity in twenty-four hours may be from thirty to fifty ounces. The stools at the height of the disease contain no natural fæcal matter, but consist entirely of mucus, serum, blood, and pus, in varying proportions, with detritus, and perhaps shreds of necrosed mucous membrane. In the early stages the discharges consist of yellowish transparent mucus, with small lumps or streaks of blood; in later stages blood is more abundant, in clots or lumps, floating in a red serous fluid. Sometimes pure blood is passed, either from early congestion of the mucous membrane, or from ulceration of the walls of the vessels: pus may be present alone, or a blackish or brownish-red offensive slimy fluid, containing portions of tissue that have sloughed away. Sometimes the stools contain curious tough masses of mucus, like frog's spawn, or boiled sago, which Heubner thinks have been formed by the coagulation of mucus in some of the ulcerated depressions of the diseased mucous membrane. The odour of the motions in the advanced stages of the disease is thought by some to be peculiar to dysentery. Two other constant symptoms are—*tormina*, or griping abdominal pains, and *tenesmus*, or the painful straining or desire to go to stool, which frequently results in no evacuation, or at most in the small quantities referred to; this is accompanied by burning pain in the rectum and anus. Frequent micturition or strangury is sometimes associated with it. These severe local conditions soon react upon the general health. The patient rapidly loses flesh and strength, the face is pale, sallow, or tinged with yellow, the tongue is covered with a thin fur, there is a moderate fever, the temperature rising to 101° or 102°, with headache, dizziness, loss of appetite, and much thirst. In milder cases the symptoms abate after eight or ten days; the pain and

scarcely perceptible pulse.
symptom in most cases, becomes
graver forms. The urine is scant;
commonly albuminous.

Other cases pass into a condit
stools are sometimes almost natural
mixtures of mucus, pus, and blood,
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severity for months or years. The p
but may with judicious treatment re
of exhaustion, or as the result of su
abscess, peritonitis from perforation
from contraction of a cicatrix.

Pathological Anatomy.—Dysente
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odour. Such patches may be quite small, or some inches in diameter. When cut into, they yield a sanious liquid, mixed perhaps with pus; and they are found to involve the submucous tissue extensively, and in some cases the muscular coat, and even the peritoneal covering. The bowel thus affected often contains a brownish liquid of gangrenous odour, sometimes mixed with blood. When the sloughs separate they leave ulcers with undermined edges, which in favourable cases cicatrize. The mucous membrane between the sloughs or ulcers is hyperæmic and infiltrated with serum and blood. The conversion of the mucous membrane and submucosa into a rigid homogeneous tissue as an acute process previous to gangrene (Heubner's *diphtheritic* form) is said by Dr. Davidson to be only of occasional occurrence in the tropics.

In the dysentery associated with amœbæ described by Councilman and Lafleur as occurring at Baltimore, the lesions were œdematous thickening of all the coats of the intestines, especially of the submucosa, gelatinous softening and suppuration of the latter, and formation and extension of ulcers by sloughing of the mucosa over it.

The parts of the bowel liable to be diseased in dysentery are the rectum, colon, and cæcum; and the disease extends in some cases beyond the ileo-cæcal valve to the ileum. The rectum, cæcum, or the lower part of the sigmoid may be affected alone. The disease is often advanced at one part, while still only commencing in another. The catarrhal form is curable, and even from the diphtheritic or gangrenous form recovery is possible if it is not too extensive. Granulations spring up over the ulcers, and cicatrices result, which are at first below the level of the islets of retained mucous membrane; subsequently the surface becomes more uniform. After extensive sloughing, the cicatrices may present bands, or cords, or contractions, which afterwards may interfere with the passage of the fæces. Besides the changes in the lower bowel, there may be some catarrh of the stomach and small intestines. The mesenteric glands are swollen and reddened. The liver is swollen and hyperæmic; the spleen, as a rule, is small. The body generally is anæmic and wasted. Persistent suppuration of the dysenteric ulcers and of the sinuses left by submucous abscesses is the cause of chronic dysentery.

Complications.—One of the most important complications is abscess of the liver, which is due to organisms conveyed by the portal vein from the diseased bowel. This is sometimes followed by an abscess at the base of the right lung; and in amœbic dysentery the amœbæ are found in both hepatic and pulmonary abscesses. Perforation of a dysenteric ulcer with peritonitis occasionally happens.

Sequelæ.—An arthritis may be mentioned, in which several joints become painful at the same time, but are neither very tender nor much swollen: there is moderate fever, and the trouble subsides in three or four weeks.

Diagnosis.—This is determined by the character of the stools, with the associated tenesmus and griping; but the two latter symptoms may be absent, and a diagnosis may have to rest on a careful examination of the fæcal discharges. Local diseases of the rectum alone may give rise to somewhat similar conditions. Patients with cancer of the rectum have a good deal of straining, and pass mucus streaked or tinged with blood; they are mostly elderly people, and a rectal examination quickly decides the question. The passage of blood in intussusception may also mislead. A simply overloaded rectum may give rise to frequent evacuations of a little mucus; but more liquid discharges of reddish serous fluid, containing lumps of mucus, with blood and pus, are characteristic of dysentery. On the other hand, dysentery seems sometimes to present so few symptoms as to be scarcely appreciable even to the patient.

Prognosis.—The mortality in different epidemics has ranged batween 30 and 80 per cent. Gangrenous stools, free bleeding, severe vomiting, and indications of collapse are unfavourable; and the disease is specially fatal to infants, old people, sickly individuals, and habitual drinkers.

Treatment.—The patient should be kept warm in bed, and should have a light fluid diet, consisting of milk, or milk and lime-water, chicken-broth, sago, arrowroot, or tapioca given warm, as cold liquids are liable to stimulate the peristaltic movements of the bowel. The most valuable remedy for dysentery is ipecacuanha. It appears to have a specific action upon the bowel, and almost loses for the occasion its familiar emetic effect. This is clear from the description of our English army surgeons in India (Maclean, Ewart), who have used this drug extensively. The ipecacuanha should be given in doses of 20 to 30 (or even 60) grains, suspended in 2 drachms of syrup of orange peel and half an ounce of water. The patient should be kept lying down, and nothing should be given to drink for some time; at most a little ice may be sucked. In many cases nausea occurs; but vomiting is exceptional, and then only after an hour or two, so that the ipecacuanha is probably retained. A similar dose may be given again after eight or ten hours, or if either is rejected, it may be repeated as soon as the stomach is again quiet. Doses of 10 or 20 grains may then be given night and morning; or the larger doses may be continued twice or three times a day if the symptoms are severe. The tenesmus and tormina are sometimes at once checked or diminished, the stools soon become more feculent and natural, and the mucus and blood nd the quantity varied according to results. Dr. Leahy ommended sulphate of magnesium. He gives a drachm turated solution of sulphate of magnesium, with 10 drops te sulphuric acid, every hour or two until the motions ecome copious, feculent, and free from blood and mucus, perature has fallen, and pain and tenesmus have ceased. astringent of dilute sulphuric acid and tincture of opium afterwards used. The salts require to be given in t cases for one or two days, occasionally three, but rarely

e the lesion is in the sigmoid or rectum, some relief may ded by enemata of warm water or infusum lini, not ex- 15 ounces. Abdominal pain may be relieved by poultices. us may be sometimes relieved by opium in enema or ory. Stimulants should only be given in small doses, in cases of collapse. As the case improves, solid diet of meat and farinaceous food, but not vegetables, may be ; and iron and bitter tonics are useful. If dysentery is ated with malarial indications, quinine should be given of 10 or 20 grains. Chronic dysentery is also benefited acuanha. The milder cases, which are occasionally seen rs landing in London, are often quickly cured by 5 or 10 loses given twice or thrice daily. Severer cases require l to a warm climate free from malaria, continued rest, a y regulated diet, and tonics, such as iron, to counteract mia. It is in these cases that astringent enemata have argely used—sometimes with much success. Gallic acid, of lead, and nitrate of silver may be thus employed. Dr. nzie treated several cases at the London Hospital, in- three pints of water holding 30 to 90 grains of nitrate of n solution. The enema was, as a rule, promptly returned ;

has broken out in any place, every insanitary condition, such as uncleanliness, imperfect sewage arrangements, contaminated water supply, &c., must be dealt with. Stools should be disinfected, as well as linen, instruments, and utensils. Overcrowding should be avoided. Individually every one should be very particular about his diet, avoiding unripe fruit, indigestible food of all kinds, or those that are likely to lead to constipation. The body and limbs should be kept warm, and chills carefully guarded against.

ERYSIPELAS.

(*St. Anthony's Fire. The Rose.*)

Erysipelas is a specific contagious disease, characterised by a peculiar form of inflammation of the skin.

Ætiology.—One of the most striking features is its tendency to attack the skin in the neighbourhood of wounds, whether accidental or the result of operations; and it hence becomes one of the troubles that the surgeon has to guard against. On the other hand, it not infrequently arises, apparently, without any previous wound, and such cases are commonly placed under the care of the physicians in hospitals. The disease is, however, the same in the two instances, as shown by surgical patients catching the disease from so-called idiopathic cases, and by patients without wounds in surgical wards sometimes taking the disease when it has become prevalent amongst those with wounds. It is now believed, as first stated by Trousseau, that in all cases of erysipelas, some wound or other can be found—a scratch, or an abraded pimple, [illegible] slight lesion of the skin.

[illegible] contagious and inoculable, but it is active [illegible] distances, and the presence of a wound forms the [illegible] cause. It affects infants and people over [illegible] more frequently than others; men and women [illegible] to it. Some conditions of the individual [illegible] chronic disease of the liver and kidneys; [illegible] and malnutrition from insufficient food. [illegible], overcrowding, bad ventilation, dirt, and [illegible] act in the same way. There may be also [illegible] it often recurs in the same person; [illegible] be inheritable. The immunity con[illegible] short-lived.

[illegible] determined by obvious wound, erysi[illegible] the face, and the present descrip[illegible] region. The *incubation* of the [illegible] days--from three to six; or in some instances much longer. The *invasion* is generally by a chill or rigors, and such malaise as commonly accompanies the onset of the specific fevers—headache, anorexia, furred tongue, and general pains. Within a few hours a red, tender spot shows itself on some part of the face, the side of the nose, the inner canthus of the eye, or the external ear. It may be determined by a lesion of the skin if this exists, and it not infrequently begins at the point of junction of the skin with the mucous membrane of one of the orifices—the nose, mouth, or external ear. The spot enlarges, and the skin becomes bright red, is swollen, tender, and pits slightly on pressure. The inflammation may confine itself to one side of the face, but more often affects both, and may extend to the scalp. It spreads with varying rapidity, the advancing edge is sharply defined, thick, and raised above the surface; and small tongue-like projections can be felt under the skin in front, which is not yet reddened. The whole face may be thus covered in three, four, or five days. At the height of the disease it presents a remarkable appearance: the features are enormously swollen, of bright or dusky-red colour, the eyelids are distended so as to look like bladders, generally some muco-pus is oozing from between them; the ears are thickened and much enlarged, and the patient is absolutely unrecognisable; the scalp is also swollen, and puffy. Often blebs form upon the cheeks or eyelids, which contain yellow sero-purulent or purulent fluid, and these may burst and leave yellow scabs, which further disfigure the patient. The lymphatic glands in the neighbourhood are enlarged and tender, and they are said to be thus affected even before the beginning of obvious inflammation of the skin.

The disease is accompanied by fever, mostly very decided. The temperature generally rises early to 102° or 103°, and reaches a maximum of 104° or 105° on the third or fourth day. About the sixth day it tends to fall rather suddenly, but may remain high if the cutaneous inflammation persists, or may rise again with any fresh outbreak of the local disorder. Indeed, it is closely dependent upon the inflammation of the skin; in some cases, perhaps more often when the erysipelas is not extensive, the temperature may not rise above 102°. The pulse is quick and full, numbering 100 to 120, or more. According to Jaccoud, and this is endorsed by Zuelzer, it is common to get an apex systolic murmur, indicating mitral endocarditis; and this generally disappears during recovery. I have often listened for this in cases of facial erysipelas, but remember only once to have heard it. The tongue is covered with a thick white fur. The urine is scanty, and in many cases contains some albumen, which may be present for some days. The inflammatory condition invades also the mucous membrane; the palate, fauces, tonsils, and occasionally the laryngeal mucous membrane may be reddened and swollen, and cause difficulties in respiration and deglutition. Delirium is common in severe cases, generally of low, muttering kind; and coma may follow. While the inflammation is still advancing on one side it may begin to subside at the points first affected. This receding edge is then less well defined, graduating both in colour and elevation into the healthy skin, as contrasted with the advancing margin. The swelling, tenderness, and pitting on pressure subside in turn over the whole of the affected area; the colour fades somewhat, but mostly changes to a brownish tint; and large thick flakes of dead epidermis now begin to desquamate. This process may take some days. After erysipelas of the scalp, the hair often falls out at the same time as the skin is shed, or somewhat later.

Death takes place from exhaustion, with delirium and coma, especially in older patients, habitual drinkers, and those with chronic visceral disease. It may arise also from complications.

Complications and Sequelæ.—Abscesses may form under the skin, or the tense skin may slough, and induration or, rarely, suppuration of the lymphatic glands may ensue. The laryngeal œdema may cause asphyxia; pneumonia and pleurisy are occasional complications, and peritonitis has been recorded by Wilks. Pyæmia and meningitis have been frequently mentioned in connection with erysipelas, but they are both rare as a direct result of the erysipelas itself. The former may arise from the wound which preceded the specific inflammation; the latter has been often diagnosed on account of delirium occurring in erysipelas of the scalp, but *post-mortem* examinations have shown that there is no meningitis in those cases, unless they originate in fractures of the skull, or the inflammation spreads inwards from the orbit. Mental disturbances may follow erysipelas. I saw, with Dr. Fyson of Walthamstow, a gentleman, who was in a state of maniacal delirium with delusions, while the skin of the face was still desquamating. He recovered in about a month; and other recorded cases have been generally favourable.

Pathology.—Microscopic examination of the skin of the affected part shows that the cutis and subcutaneous tissues are swollen, œdematous, and filled with large granular leucocytes, which in the upper layers of the cutis closely surround, as well as fill, the lymphatic vessels. The spread of the disease is said to follow the course of the lymphatics, but it is partly dependent on the direction of the connective-tissue meshes, and it is often checked or stopped at lines where the skin is closely adherent to subjacent parts—as, for instance, along Poupart's ligament and the crest of the ilium.

Fehleisen's ***streptococcus erysipelatos*** can be found in the lymphatic vessels and lymph-spaces at the advancing margin of iosis.—Erythema occurs in red patches, generally two or number, much less raised, and without pronounced fever. points out that zoster of the first division of the fifth ay cause so much hyperæmia of the skin as to be mis- r erysipelas; the limitation to one-half of the forehead, appearance of vesicles in groups should readily distin- . The phlegmonous or cellulo-cutaneous erysipelas, and erysipelas or diffuse cellulitis, described by surgical seem to be different affections: the former presents more hardness and greater swelling, but no defined edge, and ıpidly to sloughing; in the latter the skin itself is not unless from sloughing of the tissue beneath.

ıosis.—This is in most cases favourable, but least so in old , and those the subject of chronic visceral disease, alco- ɔr malnutrition.

ment.—The general treatment must be of a stimulant porting character. Abundance of milk, beef-tea, mutton- nd other fluid forms of nourishing food is required, and cases alcohol, in the form of port wine or brandy, must ı. The tincture of ferric chloride has been long regarded ɔific for erysipelas. That it does not in all cases forth- ɔp the spread of the disease may be easily observed, but rate acts as a good tonic, and should be given in doses adult) of 30 or 40 minims every three or four hours. has also been recommended. Tepid or cold sponging may ted to where the fever is unusually prolonged or high. relief is given by dusting the face or other part affected owder of starch, zinc oxide, belladonna, or boric acid; and re than on the face this may be covered with a layer of ool or wadding. Lint soaked in lead lotion, or lead and ɔtion, will also give relief, but should not be used where ; any tendency to gangrene. If there is extreme tension

DIPHTHERIA.

Diphtheria (from διφθέρα, a membrane) is an acute infectious disease, the essential feature of which is a peculiar inflammation of the mucous membrane of the mouth, pharynx, nose, larynx, or other part, with the formation of a "membrane" upon the inflamed surface; or the so-called membrane may be deposited upon the abraded skin, or the surface of a wound.

Ætiology.—Diphtheria is undoubtedly contagious, being conveyed through the atmosphere immediately surrounding the patient, as well as by clothes and other objects, and by instruments used in the surgical treatment of cases. It can also, like scarlatina and typhoid fever, be distributed with the milk supply. Its power of infection is, as a rule, not so great as that of scarlatina and small-pox, or, at any rate, its diffusion in the air seems to weaken it, so that those who catch the disease are generally those who have been in close contact with the patient. On the other hand, Dr. H. Airy has brought forward evidence to show that it may be sometimes transmitted over considerable distances of country by the wind. In spite of the facts which suggest that it may arise in other ways—for instance, its origin in connection with bad drains, and in newly inhabited houses, which have been built on "made soils," and the occasional prevalence of ordinary sore throats just before an epidemic of actual diphtheria, it is probable that infection from a preceding case is the source of it in all instances. It affects both sexes and all ages, but it is especially frequent in children up to ten or twelve years of age. Besides the conditions already noted as affecting its spread, we may observe that it sometimes complicates measles and scarlatina (*post-scarlatinal diphtheria*), and that it is more frequent in rural than in urban districts, especially in the more [illegible] parts of the former.

Symptoms and Course.—The *incubation* lasts generally a [illegible] from two to five or six days. The disease, though [illegible] does not often begin in the acute way characteristic of [illegible] scarlatina, and others; there are generally malaise, loss [illegible] headache, and there may be nausea, vomiting, or [illegible] throat is soon complained of, and if the throat [illegible] both tonsils, or the palate and uvula, are seen [illegible] swollen. Within a short time one or more [illegible] white deposit form on the inflamed surface. [illegible] simultaneously on both tonsils, or on one [illegible] may come on the uvula or the arch of [illegible] these parts more than any other part [illegible] frequently appear first. On the soft palate it can generally be seen that the patch is surrounded by an areola of deep red mucous membrane. If stripped off, the "membrane" leaves a raw surface, bleeding from a few points; and within a few hours another patch forms. In some cases the membrane extends on to the fauces, and forward on to the hard palate, presenting a continuous dense layer of yellowish-white or wash-leather colour. Coincidently with the inflammation of the throat the lymphatic glands at the angle of the jaw enlarge, and they can always be felt on one or both sides, according to the lesions within. Sometimes the typical membrane is preceded by a grey mucous secretion. Gangrene occurs occasionally in the severe cases.

The temperature of diphtheria is very variable, and runs no definite course; it may rise to 103°, 104°, or 105°, but is often throughout the whole illness much lower. The pulse is rapid and feeble, and the patient soon becomes pallid, while the bodily strength is in many cases quickly prostrated. The appetite is lost, and feeding becomes difficult and painful from the condition of the throat. In a large proportion of cases, variously estimated at 30 to 60 per cent., the urine is albuminous, and this occurs, not after the illness, as in scarlatina, but during the height of the throat symptoms. In the majority of cases, the specific inflammation is limited to the tonsils, soft palate, and uvula, but it may spread to adjacent mucous membranes—those of the nose and the conjunctiva, the Eustachian tube, and the larynx and respiratory passages. If the *nose* be affected there is more or less obstruction to nasal respiration, the mucous membrane is swollen, and a muco-purulent or thin brownish mucoïd secretion runs from the nostrils, reddening or excoriating the alæ and adjacent upper lip. It may be streaked with blood, or decided epistaxis may occur.

Diphtheria of the larynx presents the symptoms of laryngitis, and the obstruction, due to the swollen mucous membrane, is increased by the presence of the diphtheritic false membrane. The first warning is often given by the occurrence of a noisy, brassy, or croupy cough, soon followed by the noisy or stridulous breathing during inspiration and expiration, which indicates that the glottis is narrowed. As obstruction increases, the supra-clavicular, supra-sternal, and intercostal spaces are sucked in with each inspiration, and in infants and young children, with soft yielding bones, the lower end of the sternum, or the three or four lower ribs, are drawn in, showing the extent to which the air is prevented access through the glottis to the lungs.

Slight degrees of obstruction may persist some days without much change, but more often the case gets progressively or rapidly worse. The face, at first flushed, with bright eyes, gets pallid, and finally livid or cyanosed. The child is restless, putting its hand to its mouth or throat, as if to remove the impediment. The cough becomes husky rather than clanging, and from time to time there may be spasmodic closure of the glottis, in which violent inspiratory efforts are made, and the cyanosis becomes extreme. In other cases the child becomes gradually drowsy and cyanotic, the skin becomes cool, drops of perspiration stand upon the forehead, and death ensues without any struggling on the part of the child.

As a rule, in these cases the diphtheritic process is not confined to the larynx: it spreads to the trachea and the bronchi, forming a continuous membrane in the former, which, in the middle-sized and smaller bronchi, is gradually changed into a simply purulent secretion. These morbid products naturally increase the difficulty of breathing, though it is not always easy to recognise their presence by physical signs. Generally a loud and stridulous noise is heard in the chest, caused by the obstruction at the glottis. It may be mixed here and there with mucous râles, and there may be patches of tubular breathing, due to the broncho-pneumonia, which is so frequent a sequel of the spread of diphtheria into the lungs.

Diphtheria sometimes begins in the larynx, causing a membranous laryngitis. Such cases were called *croup* before the term diphtheria was in use, and for a long time there were many who held out against the belief, now sufficiently confirmed, that in nearly all cases they are of diphtherial origin (*see* Laryngitis).

Diphtheria sometimes spreads beyond the respiratory mucous membranes, when these are already affected; or from other sources of contagion membranes may form on the conjunctiva, on the mucous membrane of the female genitals, or on open wounds, such as the tracheotomy wound so often required.

In the simply pharyngeal cases, death takes place by asthenia and cardiac failure, sometimes with extraordinary suddenness. Occasionally, dilatation of the heart can be recognised by physical signs, and the pulse becomes quick, feeble, and irregular. It may thus happen on the second, third, or fourth day, or later. Sloughing of the tonsil or pneumonia may precede death.

In the laryngeal cases, death takes place from increasing asphyxia, or from an attack of spasm of the glottis; but if laryngeal obstruction has been obviated by intubation or tracheotomy, blocking of the smaller tubes, or broncho-pneumonia, may bring about a fatal result, or the patient may die from asthenia, as in the purely pharyngeal cases.

Complications and Sequelæ.—Complications are chiefly the extension of the disease to different parts, which have been described. Pleurisy may accompany the pneumonia. Albuminuria is rarely more than a symptom, but occasionally a definite nephritis may persist or occur as a sequela. The lymphatic glands may suppurate or slough.

The most important sequela of diphtheria is an affection of the nervous system, known as *dipththeritic paralysis.* This shows itself first in the soft palate. Some days, or a week, or several weeks, after apparent recovery, the child is noticed to speak with a nasal, twanging voice, and when it swallows liquids a small quantity is apt to be regurgitated through the nose. These defects are due to paralysis of the soft palate, which fails to shut off the mouth from the nose, as it should during speaking and swallowing. Shortly after this the child is noticed to be weak in the legs, and unable to walk any distance, or the knees give way on standing for a short time. The knee-jerk is lost quite early. In older children and in adults failure of accommodation of the eye for near objects is often noticed, due to paralysis of the ciliary muscle; and the extrinsic muscle of the eye may be also affected, producing strabismus or squint. In many cases the paralysis does not proceed beyond this stage, but in the course of a few weeks recovers completely. In others, the muscular system throughout the body may be affected. The patient lies motionless in bed, respiration is rendered difficult from paralysis of the intercostal muscles or diaphragm, and food given by the mouth is rejected, from inability to swallow it. The laryngeal muscles are also sometimes affected—one, or many, or all of them. Thus there may be paralysis of one cord, or paralysis of the abductors, or paralysis of all the muscles, with cadaveric position of the cords. The voice in the last case will be lost completely, and variously modified in other cases (*see* Paralysis of the Larynx).

Sensory symptoms may occur; but in children they are frequently not detected. They consist in a feeling of numbness, or formication, or distinct anæsthesia, especially in the extremities. Sometimes the muscles or the nerve trunks are tender on pressure. In severe cases electrical reactions are diminished, and some muscular atrophy ensues. Recovery generally takes place within three or four months, and the paralysis rarely, if ever, becomes chronic; but death results sometimes from paralysis of the diaphragm, and from gradually increasing accumulation of secretion in the bronchial tubes.

Pathology.—The inflammatory change which is characteristic of diphtheria is the formation of a membrane, which is separable with more or less ease from the affected surface. At the same time, as already stated, this may be preceded by a merely catarrhal stage, with the secretion of mucus; and it may go on to gangrene. The membrane is formed in two ways. In one form, a fluid exudation takes place upon the surface of the mucous membrane, there coagulating to form a membrane. In the other variety, the epithelial cells and the sub-epithelial structures are infiltrated with fluid from the vessels, and then coagulate and necrose, so as to form a greyish or white membrane. The seat of the inflammation in diphtheria has some influence upon the form which it takes. Thus, in the throat the epithelial and sub-epithelial tissues are more often affected: whereas in the mucous membrane of the trachea and bronchi of the same case the exudation is mostly upon the surface.

In the smaller bronchi the exudation becomes simply purulent; the lungs often present lobular pneumonia, with occasional hæmorrhages. The heart may be unaffected, or in cases where its failure has been a cause of death, its substance may be pale, soft, and friable, with extravasation of blood and fatty degeneration of the muscular fibres. The kidneys present changes similar to those following scarlatina—that is, they are in a stage of moderate acute nephritis, the tubes being distended with swollen, opaque, and granular renal cells.

The specific micro-organism of diphtheria is the bacillus described by Klebs and Loeffler, of about the same size as the tubercle bacillus. Two forms occur, a *long* bacillus, slightly curved and generally clubbed at one or other end, and a *short* bacillus also not uniform in thickness; but it is not clear that they are specifically distinct. They are found for the most part in the diphtheritic membrane, in its deeper layers: but may be present in small numbers, in the lymphatic glands, and in the liver, spleen, or kidneys. Their influence in causing disease is no doubt exerted through toxins, as in other instances. It is by the operation o these toxins that the paralytic phenomena of diphtheria ar explained: the clinical conditions of which are closely similar t those of alcoholic peripheral neuritis (*see* p. 180). Thus the peripheral nerve trunks have been found extensively degenerated b Mendel and others; and Sidney Martin has shown the pathological connection by injecting the chemical products of diphtheriti lesions into the veins of animals, and thus producing the sam degeneration of the nerve trunks.

The toxins of diphtheria also cause fatty degeneration of th heart, nephritis, and other lesions in the glands and spleen Streptococci and staphylococci (pyococci) are often present in th superficial layers of the diphtheritic membrane, and may sometimes lead to secondary suppurative lesions.

Diagnosis.—In early stages of a disease suspected of bein diphtheria, the diagnosis can only be positively established b the bacteriological cultivation of the Klebs-Loeffler bacillus from the secretions of the affected part. If this is not possible, attention should be paid to the following points. The presence of bright white patch, with an inflamed areola upon the uvula or sof palate, is generally distinctive of diphtheria, but some difficult may occur when the tonsil alone is affected. In *follicular tonsil*

mucoid, and often yellowish secretion ; and the occurrence
inite white patch would be generally regarded as proof of
licating diphtheria. In the earliest stages of throat in-
tion it may be impossible to say, unless by cultivations,
e rash of the one disease or the membrane of the other is
As to the diagnosis between diphtheria and *croup* it must
embered that an apparently spontaneous, or catarrhal,
laryngitis occurring in a child is most often diphtheritic.
e are patches in the throat, if there is albuminuria, if the
tis has been caught by contagion or transmits disease to
, and if it is followed (but this rarely happens) by para-
mptoms, it is diphtheritic.

theritic paralysis is distinguished by the nasal voice, the
of liquids through the nose, the loss of visual accommoda-
r near objects, the weakness of the lower extremities, and
ence of knee-jerk. In more advanced cases the friends
istake dysphagia for vomiting, and may misinform the
man.

osis.—Diphtheria is a serious and often fatal disease, but
ely pharyngeal cases are more often mild than is generally
Extensive formation of membrane, spread of the disease
nose, rapid failure of strength, and feeble pulse are of
rable prognosis, and the implication of the respiratory
s constitutes a special danger, which is only partially
l when tracheotomy has been performed. Death may occur
antitoxin) within four days of tracheotomy from pneu-
Paralysis is recovered from in the great majority of cases ;
y be fatal through failure of the intercostal muscles or
gm, or by inanition from constant rejection of food.

ment.—The principle involved in the production of im-
from certain infectious diseases (*see* Immunity) has also
pplied in the treatment of a limited number of which

[illegible] gradually increasing doses or successive injections of increasing quantities of the [illegible] of the diphtheria-bacillus, [illegible] of the [illegible]. When the animal is at length [illegible] of the diphtheria poison, its blood-serum [illegible] the power of neutralizing the influence of diphtheria [illegible], and hence it appears that the serum contains [illegible] which antagonises the [illegible] of the diphtheria-bacillus.

The serum is standardised by experiment upon animals. The [illegible] is the amount which when mixed with [illegible] protects a guinea-pig of [illegible] weight from death within four days. The initial [illegible] 1,000 units, according to the severity of the disease, and [illegible] units may be contained within [illegible] of the serum. The dose may be repeated at intervals of [illegible] hours during the next two days, and must be estimated by the intensity of the disease, and not by the age of the patient. The injection should be made under the skin of the flank with antiseptic precautions. An effect is very often observed in a few hours, either in the fall of the temperature or at least in the arrest of the progress of the symptoms. An [illegible] also sometimes follows the injection.

Treatment in general must be supporting and stimulating. The patient should be confined to bed, and good nourishing liquid food given in small quantities frequently. The fever is not often so high as to require special attention. Tonics, such as ferric chloride and quinine, or the combination of cinchona and [illegible], constitute the chief part of the internal medication. Where, with a failing heart and feeble pulse, a few drops of tincture of digitalis or liquor strychninæ should be [illegible]. [illegible] wine and brandy will be early required, and in severe cases must be freely given.

Local remedies are applied partly as palliatives, partly as antiseptics or parasiticides. They are applied either to the membrane itself or to the raw surface after the membrane has been removed by forceps. Among these, nitrate of silver and hydrochloric acid, carbolic acid, and tincture of iodine may be mentioned; and pepsin, papain, and [illegible] solutions have been used as solvents of the membrane itself. But it is undesirable to remove the membrane forcibly, or to apply powerful caustics. The more common applications are disinfectants and astringents, which, if they have no specific influence on the lesion, may at least give tone to the vessels of the part, moderate the inflammation, and prevent putrefaction. Lotions of permanganate of potash (2 gr. to ℥j), chlorine water, the liquor sodæ chlorinatæ of the U.S. Pharmacopœia, the tincture of ferric chloride (ʒss to ℥j), carbolic

t this drug must not be forgotten.
ı the nasal mucous membrane is involved local treatment
inly necessary for the removal of the offensive and irri-
ecretions. The nostrils should be syringed with dilute
tant solutions, such as potassium permanganate and car-
d; or these may be administered by the nasal douche.
ı the larynx is attacked the patient should be subjected
tmosphere saturated with moisture. In a small room it
sufficient to use a bronchitis kettle, the steam from which
the room. In a hospital the child is commonly placed in
m-tent." Much relief is also sometimes given by a hot
If improvement is not apparent in a few hours intubation
eotomy should be performed, and this must be done at once
is sucking-in of the chest, if the patient is drowsy,
ing cyanosed, or if the forehead is cold and clammy. The
ity of success is greater the earlier a tube is introduced
larynx or trachea; and if a case is seen from the first
e indications of carbonisation should be anticipated, and
ation should be done while the child is strong and of good
Nevertheless, success sometimes follows under most ad-
rcumstances; and especially it may be noted that the
e of pneumonia should not deter one even from opening
hea. The question which of the two operations is prefer-
till *sub judice*. Intubation has been successful in nume-
ses—it has the advantage of being bloodless, and, if
actory or ineffectual, it does not prevent the subsequent
ance of tracheotomy. Nearly always some improvement
n operation; the child breathes freely and deeply and
ranquilly; but it is impossible to say the patient is safe
ur or five days have elapsed. Often within thirty-six or
ght hours the exudation has spread to the smaller bronchi,
has increased and death occurs; or in a somewhat

bicarbonate (20 grains to 1 ounce). Probably the moisture has been efficacious as the solvent. The tracheotomy tube may often be removed after four or five days.

Diphtheritic paralysis generally passes off in from two to four months. Rest, tonics, and electricity are desirable. In the more severe cases, where swallowing becomes difficult, feeding by the nasal tube, or by nutrient enemata or suppositories, may be necessary. Diaphragmatic paralysis should be treated by galvanism, one pole being applied over the phrenic in the neck, the other to the epigastrium. Oxygen gas may be inhaled at intervals, and ammonium carbonate should be given in frequent doses—*e.g.*, for a child 1 to 3 grains every two hours, to relieve the accumulation of mucus in the chest.

CHOLERA.

(*Asiatic Cholera.*)

Cholera is an acute disease, of which the principal features are the profuse discharge of watery evacuations from the bowels, vomiting, collapse, cramps in the calves and feet, and suppression of urine. Two forms have been described: Cholera nostras vel Europæa, and Cholera Asiatica vel Indica. The former appears to be, in the majority of cases, an intense gastro-intestinal catarrh, occurring sporadically, brought on by particular kinds of food, or food in particular conditions of decomposition, &c. (*see* Enteritis); whereas the latter, now commonly spoken of as true cholera, is undoubtedly a specific disease, communicable from man to man, and occurring in epidemics, which have hitherto been always traceable to the home of cholera in India. The extraordinary fatality of cholera epidemics has made of each occurrence in different countries an historical event; and the spread of the disease can be traced from country to country during the best part of the present century. There were severe epidemics of this disease in the end of the eighteenth century in Pondicherry, French India, and Madras; but the history of cholera generally dates from the fatal epidemic in 1817 at Jessora, where 10,000 deaths took place in two months' time. The disease continued to be rife during the next two or three years. It spread to the coast of the Persian Gulf in 1821; in 1823 to Burmah, China, and Orenburg in Europe; in 1830 it was felt in Persia and in the large towns of Russia; in 1831 in Arabia, Egypt, Constantinople, Poland, Silesia, and Prussia. In 1832 North-west Germany and the British Isles were attacked. From England it crossed to France, and from the Atlantic seaboard to the New World, where it appeared in New York, Canada, and New Orleans. In 1833 it reached Mexico, and thence seems to have been brought back to Portugal, Spain, and Italy. From Italy it crossed to North Africa in 1835. The following two years it was again in Italy, and spread northwards into Germany. In 1845 it was severe in India; it attacked Russia in 1846, Constantinople in 1847, Prussia in 1848, and London in 1849. England since then has suffered two epidemics: a very severe one in 1854, a milder and more restricted one in 1866.

Ætiology.—Cholera closely resembles enteric fever in the way in which it is conveyed—that is, there is rarely, if ever, direct contagion from man to man, as in scarlatina and small-pox; but the *materies morbi* is present in the dejections, and it is by means of these contaminating water used for drinking, cooking, or washing, that its entrance into other individuals is effected. It is sufficient here to refer to the historical Broad Street Pump, which in 1854 was the cause of a severe epidemic. The epidemic was stayed when the pump was padlocked; and it was subsequently shown that the discharges from a patient, who had contracted cholera elsewhere, had found an entrance into the soil from which the pump-water was drawn. In 1866 the epidemic in London mainly affected the eastern portion, supplied with water by the East London Waterworks. A reservoir belonging to the water company was found to have been contaminated with sewage materials, filtering through the soil from a leaking sewer pipe; and this sewage was infected with cholera by the dejecta of a patient previous to the general outbreak. The spread of the disease in India is often determined more or less by the direction of the rivers.

Although in India the Sepoys living on the lower levels are less susceptible to it than the hill tribes, in epidemics affecting Europeans and others it makes but little distinction of sex, age, or condition. It is rare, but occasionally occurs, at high altitudes. Summer and autumn are the most favourable seasons; and it is promoted by alternating dry and wet weather, and checked by protracted drought or excessive rains. Its spread is stopped by cold, but it will survive the winter and break out again in the spring or summer. Individual predisposition is shown especially in poverty, malnutrition, and chronic alcoholism; and the bad influence of excesses of diet has been noticed in the frequency with which people have been attacked on Monday or Tuesday, obviously as a result of the Sunday's feast.

As a rule, one attack of cholera is protective against a second.

Course and Symptoms.—The *incubation* is mostly from two to three days, but may be a week or two; exceptionally it is less than two days. There is frequently a *prodromal* or *premonitory* stage of diarrhœa, or in the absence of diarrhœa the patient is depressed and uncomfortable, and complains of headache, vertigo, noises in the head, or oppression at the epigastrium; and this stage lasts from one to two or three days. Then the patient is seized with violent diarrhœa, and the discharges soon lose all biliary colouring matter, and look like whey, or water in which rice has been boiled (*rice-water stools*). These are neutral or slightly alkaline, of sp. gr. 1006 to 1013, containing sodium chloride, albumen, and mucin. If allowed to stand they deposit a finely granular whitish-grey sediment, consisting of epithelium, leucocytes, shreds of tissue, crystals of ammonio-magnesian phosphate, bacteria, comma-bacilli, threads of algæ, and blood-corpuscles. Sometimes the stools have a pinkish tinge from admixture of blood. The purging is accompanied by borborygmi and gurgling, but by little pain or griping. After one or more hours of purging, vomiting sets in; at first food is ejected, then a whey-like fluid, like the intestinal discharges. The vomiting is easy, often a mere regurgitation. The patient suffers from anorexia and thirst, the tongue is white and may become dry, and the epigastrium is sensitive to pressure. About the same time, in most cases, there are severe and extremely painful cramps in the calves of the legs, in the feet, and less often in the hands and trunk. Soon the patient sinks into *collapse*—the *algide* stage. The surface of the body becomes cold and livid, the hands, feet, face, and nose are pinched and blue, the eyes are sunken, and the breath is cold; the axillary temperature falls 4° or 5° below the normal, while in the mouth it may be even lower (79° to 88°, Goodeve). On the other hand, in the rectum and vagina it has been found to be 102° or 104° during cholera collapse. In severe and fatal cases the eyes become dry and the cornea cloudy. The pulse is small, thready, almost imperceptible, numbering from 90 to 100. Respirations are short and quick, from 30 to 40 in the minute. There is great muscular prostration, but the patient is restless, throwing his limbs about; the voice is hoarse, or sinks to a whisper (*vox cholerica*), or only the lips are moved in the attempt to speak. Purging often ceases during collapse, but vomiting continues. The urine becomes scanty, and is often entirely suppressed—a condition which may begin quite early, and last thirty-six or forty-eight hours: it is probably only a result of the failing circulation. The patient generally retains complete consciousness, though lying apathetic and indifferent, except when aroused by the pain of cramp. This stage begins six or seven hours after the first symptoms, and lasts twelve or twenty-four hours, when the patient may die without rallying. Sometimes, indeed, the state of collapse may set in at once, and the patient may sink before any purging has taken place.

In cases that survive the collapse there is a gradual rise of temperature, the skin begins to regain its natural colour, and loses its shrunken appearance, the cramp and restlessness cease,

us, which commonly begins on the hands, backs of the
s and feet, and spreads to the trunk (*roseola cholerica*).
rs at the end of the first week, or in the second week,
s from two to four days. Sometimes the reaction is im-
either a relapse occurs with purging, vomiting, collapse,
n a fatal result; or diarrhœa continues, or vomiting or
ness. Another serious condition is the so-called *cholera-*
which occurs about the end of the first week. There is
ostration, with headache, flushed face, coated tongue, loss
tite, nausea, or vomiting; the bowels may be loose or
. Vertigo is frequently complained of, and the patient
drowsy or comatose. The temperature rises to 100° or
e pulse is weak and small, and there is mild delirium at
The urine is albuminous. Goodeve described rigidity of
cles, resisting attempts to open the mouth or straighten
Lebert says convulsions are rare in children, but trismus
nus are met with in adults. The condition lasts from
nine days, ending in recovery, or in death by coma. It
nonly been attributed to uræmia.

ications.—The following have been observed during the
stage, or during convalescence: bronchitis, pneumonia,
sy; diphtheritic inflammation of the pharynx and larynx,
and female genitals; parotitis; gangrene of the scrotum
is, or of the nose; opacity and ulceration of the cornea
osure during the stage of collapse; and bed-sores.

ies.—Apart from differences in the severity of the pro-
disease, the only varieties that can be described are two
orms of disease which occur during cholera epidemics,
no doubt due to the same cause. One is called *choleraic*
, the other *cholerine*. The former begins suddenly, after
to cold or some error in diet, with profuse but painless

cooling of the extremities, scanty urine, and albuminuria. Recovery is slow.

It is said that either of these varieties may pass into the true cholera.

Pathology.—Decomposition proceeds slowly in those dead of cholera, and *rigor mortis* persists a long time. The right side of the heart is often distended with blood, the lungs are engorged, and the mucous membrane of the trachea and large bronchi is congested. There is often purulent mucus in the minute bronchi, and in cases dying in late stages there may be hæmorrhagic infarcts. The *intestine* contains in earlier stages rice-water fluid, in later stages liquid of a more greenish colour. Peyer's patches and the isolated follicles are swollen and prominent, and yield on puncture a whitish-grey fluid, with fine granules and cell-nuclei. Later they become shrivelled or abraded. The *spleen* is small. The *kidneys* are large, and show proliferation of the epithelium and cloudy swelling. Later on, casts form in the tubes, the organs become paler, and fatty degeneration takes place.

Micro-organisms.—Lebert long ago formed the opinion that there must be a cholera germ; but it was only in 1884 that Koch asserted that he had discovered a bacillus, which he believed to be the real cause of cholera. This organism, familiar as the *comma-bacillus* or *vibrio* of cholera, is found in the rice-water evacuations, in the contents of the intestine after death, and in the mucous membrane of the intestine, just beneath the epithelium, but it has not been found in the blood. It is a little shorter than the tubercle-bacillus, slightly curved, somewhat thicker in the middle than at the ends, and flagellated at one or both ends. It can be cultivated in the usual media, but it has not even yet been satisfactorily proved that it is the specific cause of cholera. Similar vibrios occur in other conditions than cholera—*e.g.*, in guinea-pigs' intestines, in human saliva, and in the discharges from diarrhœa and dysentery; and, on the other hand, considerable differences are found in the vibrios obtained from different cases of cholera. The specificity of the cholera vibrio is, however, supported by two facts: one, that the serum of persons recovered from cholera is protective against inoculations of animals with cholera vibrios; the other, the successful immunisation of the human subject by cholera antitoxins.

Diagnosis.—Cholera, in its milder forms, may be exactly simulated by the severer kinds of diarrhœa from gastro-intestinal irritation and ptomaine poisoning, in which there may be pronounced collapse, prostration, scanty urine, and loss of colour in the motions. These cases are generally single or in family groups. Arsenical poisoning, producing similar symptoms, has been also mistaken for cholera.

tment.—It has been the general practice to treat with
and astringents the diarrhœa which is prevalent during
ics of cholera. These should be used in the premonitory
but they are of no good in the pronounced stages of the
itself; and excessive medication in any form may do harm,
hatever is introduced into the stomach will either be
l again, or will lie unabsorbed, from the inactive condition
circulation. Even stimulants, such as brandy-and-water,
ia, or ether, must be given in small quantities and with
. Some relief may be obtained from sucking ice, or taking
uantities of ice-cold water or of effervescing draughts.
cramps may be treated by small doses of morphia sub-
usly, or by gentle friction with chloroform or stimulating
ts. Cold compresses will relieve the præcordial distress.
ndition of collapse must be met by supplying heat by
f warm blankets and hot bottles to the feet or the sides
chest and thighs. At the London Hospital, in 1866,
atients were immersed in baths of from 98° to 104°
parent benefit. The intravenous injection of saline solu-
a composition similar to that of the plasma, or of sodium
alone (·7 per cent., or about a drachm to a pint), has
d to do good in some cases of profound collapse, but the
ment is generally of very short duration, and many regard
ctice as useless.

e stage of reaction the patient should be kept cool;
a, if it continues, may be checked by opiates or astrin-
the vomiting by effervescing draughts or by opium. Light,
us food should be given frequently in small quantities.
uch vomiting it may have to be given *per rectum*. Con-
suppression of urine will require small doses of salines, and

ments, with reference especially to water supply, drainage, and the removal of sewage, the less likely is this to take place. In all these respects cholera and typhoid fever are precisely the same. In the "Precautions against Cholera," drawn up by the late Sir G. Buchanan, when medical officer of the Local Government Board,* it is pointed out, first, that any choleraic discharge, cast without previous thorough disinfection into a cesspool or drain, has a faculty of infecting the excremental matters with which it there mingles, and probably more or less the effluvia which those matters evolve; secondly, that the infective power of choleraic discharges attaches to whatever bedding, clothing, towels, and like things have been imbued with them, and renders these things, if not thoroughly disinfected, capable of spreading the disease to places to which they are sent; and thirdly, that if by leakage or soakage from cesspools or drains, or through reckless casting out of slops and washwater, any taint of the infection gets access to wells, or other sources of drinking water, it imparts to enormous volumes of water the power of propagating the disease. The above indicates the direction in which one should act: disinfection of the discharges, and of everything soiled with them, and constant watchfulness to avoid possible contamination of water, as well as air and food supply. Cleanliness in every way helps in this respect. Quarantine, as understood on the Continent, has long been distrusted by English authorities, and the regulations issued with regard to vessels entering British ports only provide for thorough inspection by the medical officer of health, who has to see that such means are taken by disinfection of the vessel, and by the removal and detention, under proper treatment, of any that may be suffering from cholera, as will prevent its spread to healthy individuals.

Prophylactic treatment has been attempted in another form of late years—namely, by inoculation of healthy individuals with cultivations of the cholera-bacillus. M. Haffkine has elaborated a method, which has been used extensively in India, with promising results. He injects first an attenuated culture of comma-bacilli, obtained by growing them on artificial media, continually aërated; and then a stronger ("exalted") culture prepared by inoculating the peritoneal cavities of a series of guinea-pigs. As each animal dies, the fluid from its peritoneal cavity is used to inoculate the peritoneum of another, and so on until twenty or thirty animals have been employed, when the maximum virulence, estimated by the shortness of the period between inoculation and death, has been reached.

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* Fifteenth Annual Report, 1886, p. 107.

:. This is an acute febrile disease, attended by swelling of
mphatic glands in the groin or other part of the body, and
mes by carbuncles. Its history can be traced back to the
century of the Christian era, but the first great epidemic
rope, the plague of Justinian, occurred in the sixth century.
mics were frequent in the Middle Ages; but since the year
when London was devastated by the plague, these epidemics
radually become less frequent in Europe, and in the first
of the present century were confined on this continent to
st easterly portions of the Turkish Empire. Since 1841,
edom of Europe has only been interrupted by the appear-
f the plague in 1878–79, in some villages on the banks of
olga. In 1894 there was a serious epidemic in Hong
and South Eastern China; in 1897 the disease appeared in
y, and has since broken out in other parts of India.

bts are entertained as to the connection between plague
e Black Death of the fourteenth century. The latter was
uished by severe pulmonary complications, with hæmo-
and it is regarded by some as having been a distinct dis-
Máhámari, the Pali or Indian plague, and the plague of
n, in Western China, are probably only varieties of the
l plague.

logy.—The plague is a specific disease communicable from
o man, and it has generally been credited with being
contagious. But it is possible it may be transmitted by
nation of soil or water, like cholera and enteric fever. In
e its spread is favoured by all those conditions which are
rtant in the case of other fevers—namely, overcrowding,
t ventilation, accumulation of sewage and other filth about
g-houses, and the pollution of air and water supply. The
of the year and climate have an influence, but apparently
e same in all countries: very great heat seems to have

and shivering, soon followed by febrile reaction. Sometimes in this stage of invasion the patient is in a peculiar absent condition, with staggering gait and tremulous speech; or he is seized with indefinable fear and restlessness; or there may be nausea, vomiting, or diarrhœa. The fever is generally high, the temperature from 102° to 104°, or in the worst cases over 107°, the pulse from 100 to 130. The tongue, at first moist and white, becomes dry and brown, and a typhoid condition may supervene with delirium or coma, sordes on the lips and teeth, failing pulse and cold extremities. In the Volga epidemics, the urine was scanty or suppressed. After one, two, or three days' fever the local signs show themselves in the formation of glandular swellings in the groins, axillæ, or neck. Mostly only one group is swelled, and generally it is the inguinal glands that are affected. The swelling may be as large as a hen's egg, is attended with severe pain, and if the patient survives may suppurate about the seventh day. About this time also boils or carbuncles may appear, but they are not very frequent; they occur on the lower extremities, the buttocks, or the back of the neck. In the severest cases petechiæ, or larger subcutaneous hæmorrhages, appear shortly before death, either distributed generally over the body, or more marked in the neighbourhood of the enlarged glands; and there may be bleeding from the nose, lungs, stomach, or bowels. Death takes place mostly before the sixth day. In cases that recover convalescence begins from the sixth to the tenth day, but may be much protracted by suppuration of the glands.

Sequelæ.—Furuncles, pneumonia, dropsies, partial paralyses, and mental disturbances are mentioned as sequelæ.

Varieties.—Netten Radcliffe, described a *larval* or abortive form (*pestis minor*), in which the buboes appear without any fever, but either suppurate or more often subside in about fourteen days; and a very severe, or *fulminant* form (*p. siderans*), in which patients are struck down and die in a few hours. Some such cases, occurring on the banks of the Euphrates in 1873–74, were characterised by intense fever and vomiting of blood.

The mortality has always been very great, reaching 40, 50, or even 80 per cent. In the early period of an epidemic nearly every case is fatal.

Morbid Anatomy.—The enlarged lymphatic glands are found to be inflamed, red or violet in colour, soft or pulpy in consistence, and surrounded by connective tissue infiltrated with serum or blood. The internal glands in the same neighbourhood are involved; thus the pelvic glands with inguinal buboes, or the mediastinal glands with buboes of the neck. The spleen is large and dark, and there are ecchymoses of the mucous or serous membranes, and swelling of the kidneys with extravasation into their substances and into the surrounding cellular tissue. Kitasato discovered a bacillus in the blood, in the buboes, in the spleen, and other internal organs.

Treatment.—This is, at the best, unsatisfactory. Quinine and carbolic acid have been used internally in recent epidemics, and leeches and mercurial frictions have been applied locally. Beyond this it must be symptomatic—the hyperpyrexia, collapse, thirst, &c., being dealt with as in other acute specific diseases.

Prevention.—This comprises the improvement of sanitary conditions, personal and local; the isolation of any patient suffering from the disease; and the disinfection of his clothes, bedding, &c.

SYPHILIS.

Syphilis, or The Pox, is a specific infectious disease, conveyed by inoculation, and producing successively a lesion at the seat of inoculation (*primary* lesion); lesions of the skin, mucous membranes, and other parts after an interval of a month or more (*secondary* lesions); and, after one or more years, deeper lesions of the skin, bones, muscles, and viscera (*tertiary* lesions). It can be transmitted from parents to children, and then takes on forms which differ in some particulars from the disease acquired in the usual way.

ACQUIRED SYPHILIS.

Infection.—Syphilis is, as a rule, communicated during sexual intercourse, the delicate epithelium of the mucous membranes brought into contact allowing of the easy transmission of the virus. Cracks or abrasions of the mucous membrane do not seem to be necessary for the reception of the poison, though they must undoubtedly favour it. Syphilis may be also transmitted in other ways—for instance, in the act of kissing, by smoking pipes previously used by syphilitic persons, or by contact of syphilitic sores or secretions with the abraded finger of the medical man. Inoculation by sexual intercourse takes place commonly, in the male, on the glans penis or prepuce; in the female, on the vulva, labia, or vaginal mucous membrane. After inoculation there is usually a period of *incubation*, which varies from two to four or five weeks, and during which no change whatever may be observed.

Primary Lesion.—The first sign is a small red itching papule, which gradually enlarges in all directions like a flat button, and becomes very hard. The surface is dry, or scaly, or superficially ulcerated and covered with a crust of dried secretion. This condition of induration, which is most important, is reached in a week or ten days from the first appearance of the papule; and the lesion is now known as the *hard*, *indurated*, or *Hunterian chancre*. On the mucous membrane the lesion may be scarcely so well marked; it begins as a vesicle with a red base, the vesicle breaks, and forms a shallow ulcer, the floor of which becomes indurated. In the course of time, and it may be some months, the induration gradually disappears, the ulcer heals, and a patch of pigment is left behind for a while. Though this is the typical course of the primary lesion in syphilis, this poison is often produced or is present in a lesion of a different character—the *soft chancre*. It begins as a pustule, which breaks in two or three days, and forms a deep, irregular ulcer, which has no hard base, and secretes pus freely. This pus is inoculable in the neighbourhood, and such soft sores are frequently multiple, whereas the hard chancre is generally single. The soft chancres are venereal in origin, but may be produced by other causes than syphilis; nevertheless, they do sometimes contain the syphilitic poison, and all the later results of constitutional infection may then follow without the occurrence of any induration. As a further complication of this stage, it should be mentioned that hard sores may, by the irritation of caustics, be made to suppurate freely, so as to simulate soft sores. During the first stage of syphilis, the poison is transmitted to the *glands* of the groin; these become indurated, and slightly enlarged, but remain freely movable upon one another, without adhesion or reddening of the skin, and do not, as a result of the hard chancre, undergo suppuration.

Secondary Symptoms.—These are noticed from four to six weeks after the stage of induration of the chancre, and may continue to appear at any time up to twelve months. The most constant are certain eruptions on the skin (*syphilodermia*, *syphilide*), faucial inflammation, and enlargement or induration of lymphatic glands; others are febrile reaction, pains in the temples, back, or limbs, swelling of the joints, iritis, and falling of the hair.

Eruptions.—Important differences are to be noted between the early or secondary rashes, and the late or tertiary eruptions. The former are generally abundant, affecting a large part of the body, trunk, or face, and symmetrically arranged; the separate elements are more or less discrete; the lesions are superficial, do not ulcerate and leave no scars. The latter are few, scattered, unsymmetrical, often affect the deeper layers of the skin, and form considerable ulcers, which are followed by scars; sometimes even, as in lupus, scars are formed without preceding ulceration. The elementary lesions (*see* Diseases of the Skin) of early syphilitic rashes are very variable; they may be maculæ, papules, nodules, scabs, vesicles, or pustules; and the lesions are not infrequently mixed in the same cases, constituting the feature of *polymorphism*.

The most common form of "secondaries" is the *macular* syphilide, which forms dusky pink, or reddish brown spots, rather thickly grouped so as to form a mottled rash on the face, chest, back, and limbs. It is very variable in its duration, but may persist some weeks. It does not itch. Other forms are a *follicular* syphilide, consisting of small papular elevations, with a hair in the centre of each; a *papular* syphilide, with elevations, flat, or hemispherical, or more prominent still, so as to form nodules or tubercles, which come out in crops, irregularly over the whole body, or grouped in clusters; a *pustular* form, in which successive crops of pustules occur, and are often accompanied by fever, the pustules drying up into brown or greenish scabs, and leaving behind slight scars: a *squamous* form, which differs from ordinary psoriasis in the scales being less thick or shiny, in the coppery tint of the rash generally, and in the patches not being seated on the knees or elbows, or specially on the extensor surfaces; a *vesicular* and a *pigmentary* form, both of which are rare. As a special form of papule may be mentioned the mucous patches (*plaques muqueuses*), or flat warty growths which occur about the genitals, perineum, and anus, in the axillæ, groins, and under the breasts, and at the angles of the mouth—in any place, indeed, where the skin is thin and constantly moist. They are often rather extensive, with well-defined edge, moist surface, and dirty-grey secretion.

Sore Throat.—Coincidently with the rash, or even before it, the throat becomes affected; there is a diffused redness of the fauces, with enlargement and excoriation of the follicles; but the most characteristic feature is the swelling and symmetrical ulceration of the tonsils. The ulcers are often kidney-shaped, superficial, with greyish borders, painless, and not of very long duration. Sometimes, however, the tonsillar ulcers are much more persistent, extend to the soft palate and uvula, have bright red edges, and are covered with a yellowish-gray secretion, the removal of which is followed by bleeding. Other changes in the mouth in the secondary stage are white spots, like those produced by the application of nitrate of silver, mucous patches on the tongue or cheeks, bald patches on the tongue from the destruction of the papillæ, or enlargement of the tongue, which is of a bright red colour, with hypertrophied papillæ, or irregular prominences, and deep sulci between them: this last condition is aggravated, or in part caused, by excessive smoking.

The *lymphatic glands* are enlarged, especially in the groins, above the inner condyles, and at the back of the neck. The *fever* of constitutional syphilis is often entirely absent, or it is represented by no more than a slight malaise or indisposition preceding or during the outbreak of the skin. In a small number of cases there is very distinct intermittent or remittent pyrexia, the temperature highest in the evening; and it may last for some weeks. The *hair* may come off in considerable quantities from the scalp, as well as the surface of the body and limbs; but it is not common for complete baldness to occur. The nails are sometimes affected in their nutrition also. The *periostitis* of secondary syphilis is slight; pains and tenderness are felt in the tibiæ, skull-bones, or clavicles, but they are of short duration, and nodes do not generally form as in the tertiary stage. Transitory swelling of the joints also takes place.

The most common affection of the eye is *iritis;* it usually affects one eye before the other; the symptoms are photophobia and pain, with ciliary congestion, irregular pupil, obscured iris, and, in severer cases, nodules of rust-coloured lymph and blocking of the pupils. Iritis occurs from three to six months after contagion; at a later period still, but within the limit of secondary symptoms, there may be diffuse retinitis or choroiditis.

For a long time the *nervous system* was regarded as exempt from the attacks of the syphilitic virus until the later, or tertiary, period; but there is much evidence to show that various nervous affections, especially myelitis, are apt to occur within a few months or a year of infection, and thus to fall within the category of "secondary" results. Such disorders show a considerable proportion of recoveries under antisyphilitic treatment; but, pathologically, they present either inflammatory changes not peculiar to syphilis, or at most arterial changes of a specific character.

Some other lesions occur at a time which is intermediate between the second and tertiary periods, such as scaly or peeling patches on the palms of the hands (*psoriasis palmaris*); enlargement of the testis with perhaps nodular deposit in the epididymis; choroiditis and retinitis; and transitory visceral changes, not due to gumma—for instance, enlargement or tenderness of the liver and spleen, with failure in the blood-making process, slight and temporary albuminuria, and symptoms of impending lung mischief. And, indeed, no hard-and-fast line can be drawn between the end of the secondary and the beginning of the tertiary stage.

Tertiary or Late Symptoms.—These appear first from one to two years after contagion, and may continue to break out for ten or fifteen years, or more. The characteristic lesions are certain late eruptions on the skin, periostitis and nodes on the bones, and growths in the subcutaneous tissue, muscles, meninges of the brain, spinal cord, and viscera, especially the liver, spleen, and testis.

Late Syphilides.—The special features of these have been already mentioned (p. 126). As in the early rashes, there may be considerable variety, and the rash may consist of maculæ or scaly patches. But the most characteristic is a dusky red, infiltrated patch, forming a circle or broad band curved in a half-circle or horseshoe; part of the surface is covered with a brown or greenish scab, beneath which are deep ulcers with sharply cut edges. The lesion spreads in serpiginous lines by the formation of fresh infiltrations or nodules, which in turn ulcerate, while the old ulcers heal and leave scars surrounded by deeply pigmented skin. Sometimes such nodules will subside and leave scars even without ulceration, and altogether there is a general resemblance to lupus.* Ultimately large, irregular patches, of several inches in diameter, may form, and they are frequent on the knee, thigh, shoulder, forearm, face, and neck. Sometimes much deeper infiltrations of the subcutaneous tissues occur.

The growths in the viscera and other parts, which are so characteristic of the later stages of syphilis, are known as *gummata*. They consist of a substance like granulation-tissue, with a varying proportion of cells. In early stages they are greyish, gelatinous, transparent; but the cells undergo fatty change, and caseation takes place, so that the centre becomes yellow, and the circumference develops into fibrous tissue, which contracts like that of a scar. Sometimes gummata break down completely, and suppuration, with destruction of the tissue in which they are situated, takes place; thus, caries and necrosis not infrequently follow nodes on the bones. In the liver, gummata form large, more or less uniform, yellow nodules; or a yellow caseous mass lies in the centre of a fibrous cicatrix, or nothing is left but the fibrous cicatrix, with consequent depression and puckering of the organ. In the testis, gummata also occur; but this organ is often enlarged by effusion into its substance generally, and may afterwards atrophy from the formation of a dense fibrous tissue without any local nodular growth. For the clinical results of these lesions, the reader is referred to the diseases of different organs. It will be sufficient here to say that gummatous periostitis, or nodes, occur especially along the anterior surface of the tibia, on the frontal and parietal bones, and on the clavicles. The patient suffers from pains which are worse at night, and there may be found, on the affected part, flat, round prominences, from half an inch to an inch in diameter, soft, or even fluctuating, and very tender. This is not necessarily a sign of pus being present, as quite distinctly fluctuating nodes may entirely disappear under treatment. Several disorders of the nervous system are referable to syphilis; some, like hemiplegia, are due to syphilitic disease of the arteries (*obliterative endarteritis*); others, like nerve paralyses

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* The name "syphilitic lupus" is in use, but it does not seem desirable that any syphilodermia should be named directly after the skin diseases which are not syphilitic. Macular, papular, pustular, ulcerating syphilide, are better names than syphilitic roseola, lichen, acne, or lupus.

during the outbreak of the skin. In [illegible] of the brain
there is very distinct intermittent or remitt[illegible] through the
perature highest in the evening; and it [illegible] underlie them
The *hair* may come off in considerable [illegible] from what occa-
as well as the surface of the body and [illegible] are possibly toxic
for complete baldness to occur. The [illegible] ophthalmoplegia,
in their nutrition also. The [illegible] membranes are
slight; pains and tenderness are [illegible] such as we see
clavicles, but they are of short [illegible] palate, with adhesion
form as in the tertiary stage. T[illegible] trachea, bronchus or
also takes place. [illegible] passages. Lastly,

The most common affectio[illegible] degeneration,
affects one eye before the other [illegible]; and the disease is
and pain, with ciliary conc[illegible] in the production of
and, in severer cases, nodul[illegible]
of the pupils. Iritis occur[illegible] to which the disease
gion; at a later period [illegible] influenced by treat-
symptoms, there may be [illegible] stages may entirely

For a long time [illegible]; and the secondary
from the attacks of the [illegible] extremely mild by diligent
period; but there is [illegible] recognised. The disease
affections, especially [illegible] two stages, but in late
months or a year [illegible] tumours, and bring
of "secondary" [illegible] function, especially
portion of recov[illegible] results from syphilitic
logically, they [illegible] rectal stenosis, peri-
to syphilis, or [illegible] disease of the liver,

Some other [illegible]
between the [illegible] syphilis is still unknown,
patches on th[illegible] infection which it is
ment of the [illegible] secondary lesions are
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rious occurrence of miscarriages; but it must not be for-
how frequent are miscarriages under quite different
tances, and how one miscarriage, from whatever cause,
to be followed by others.

h should be made for scars of the original sores on the
men, for scars of tertiary lesions on the skin and in the
for nodes on the tibiæ and skull, for hardness or atrophy
testes, and for evidences of lardaceous disease, in the size
ver and spleen, and in the existence of albuminuria.

ment.—Mercury is the drug which by universal consent
d to have the most powerful influence in the treatment
lis. If given with sufficient persistence from the first,
y diminishes the induration of the primary chancre, and
s off, or considerably modifies, the secondary lesions.
y, it lessens the severity and frequency of the outbreaks
ry symptoms, if it is continued for some time after the
of the secondary lesions. The most desirable method
e the drug in such doses that it may be continued day
y, and week after week, without inconvenience to the
–that is, in short, it must not be allowed to cause
n. It may be given in many forms, but, for the above
the milder are preferable. The more usual are the
ide in doses of $\frac{1}{16}$ to $\frac{1}{12}$ grain (60 to 80 minims of the
hree or four times a day, and hydrargyrum cum cretâ
wder) in doses of 1 or 2 grains with the same frequency.
inson prefers 1 grain of grey powder, with a grain of
powder, if necessary; to be given every six, four, three,
ours. The drug may also be administered by the skin
n). A small quantity (half a drachm) of unguentum
ri is rubbed into the thin skin of the axillæ and groins
ch axilla and each groin being successively used for this

injected into the gluteus maximus once a week (Bloxam): or the following may be employed:—One grain of red iodide of mercury in 64 minims of distilled water, with sufficient sodium iodide to dissolve it: dose 2 to 6 minims.

During the use of mercury the patient should abstain from smoking and from stimulants, and live in every way as healthy a life as possible.

The mercurial treatment, whichever form be chosen, should be always, if possible, continued in the primary or secondary stages, and it is frequently of value in tertiary lesions; but here another drug takes the most important position—namely, *iodide of potassium*. Under its use the most serious and alarming nervous symptoms, due to syphilitic lesions, rapidly subside, ulcerating skin lesions quickly heal, and syphilitic bone pains and nodes disappear. Five or seven grains three times a day are often sufficient, but in serious cases it may require to be pushed to ½-drachm or drachm doses three times daily; or, in a still worse case, it may be used as recommended by an American physician —that is, 20 grains are given in a little milk every two hours through the whole day and night. The advantage of this, no doubt, lies in the thorough saturation of the system; otherwise, as the salt passes away rapidly by the kidneys, the amount in the body may fall very low in a long night interval. If iodide, in any dose, causes coryza, it should be taken much diluted—*e.g.*, in half a tumblerful of water; arsenic may be added if it causes eruptions (*see* Medicinal Eruptions). General tonics, good food, and sea air are desirable if it causes much depression, or the iodide of sodium may be given instead in corresponding doses. If these fail, recourse may be had to mercury, either alone or with a tolerable dose of potassium iodide. The destruction of the primary sore is probably useless to prevent infection. To soft sores iodoform is a good application, or healthy action may be set up by the use of the acid nitrate of mercury. Iodoform is also of value as ointment or powder to ulcerating skin lesions, and by means of insufflation to ulcerating tonsils.

CONGENITAL SYPHILIS.

Children born of parents suffering from syphilis in the first or second stages may themselves be infected with the disease. As a rule, this transmission does not take place in the tertiary stage. The disease may be taken from the father alone, the mother being healthy (sperm-inheritance, or paternal conception inheritance); or from the mother alone, the father being healthy (germ-inheritance, or maternal conception inheritance); or from both father and mother; or, lastly, the mother may acquire syphilis after conception, and convey it to the fœtus *in utero* through the placental blood (pregnancy inheritance). According to Hutchinson, the symptoms of the child's disease are the same whether the disease has been acquired in one or other of these ways; whether the disease of the parent was in the primary or secondary stage; and the symptoms are not necessarily more severe when they are derived from both parents than when they come from one alone. Further, Hutchinson denies the universality of the law by which the children born at the earliest period of the parent's disease are more severely affected than those born afterwards. As a fact, the transmission is irregular—one child may be badly affected, another not at all. Experience of congenital syphilis has, however, brought out a law to which the exceptions are very rare—namely, that if a child inherits syphilis from its father, the mother being originally healthy, the syphilitic infant may infect a wet nurse—*e.g.*, causing a chancre on the nipple—but will not infect its own mother; thereby showing that the mother is in some way protected against infection, though she may manifest no lesions whatever of a primary or secondary kind. This is called *Colles' law.*

Death of the Fœtus.—One effect of syphilis in the parent is the early death of the fœtus, with resulting miscarriage or premature birth—so frequent that the fact of miscarriages having occurred in the history of a married woman may be important evidence as to syphilis in herself or her husband. It is not so easy to say the exact cause of the fœtal death—whether from the immediate effects of the syphilitic virus or from some disease of the placenta. Hard yellow masses have been found in this structure, and some peculiar changes in the villi; but their significance is as yet uncertain. On the other hand, the fœtus not infrequently presents lesions of the bones, viscera, and skin, which show that it may be profoundly diseased. In the bones a change takes place at the line of junction of the epiphysial cartilages and the shaft, allied to that of rickets, and described as osteochondritis. The cartilage may be separated from the bone by soft granulation-tissue or pus.

Early Symptoms.—Such a change is sometimes present in children born alive, the principal epiphyses being separated from their bones, and the limbs consequently lying useless, so as to give the appearance of paralysis. Occasionally also the child is born with a bullous eruption on the skin (*Pemphigus neonatorum*), or the rash comes out very soon after birth. But in a large majority of cases the child is born not only alive, but healthy, fat, and plump, and remains so for three or four weeks after birth. Then it acquires a nasal catarrh, causing the symptom commonly known as *snuffles*, with a discharge—at first thin and serous, afterwards thicker, purulent, and drying up into crusts, which obstruct the nostrils, so that sucking is difficult. At the same time a *rash* appears, most commonly on the buttocks and adjacent parts of the thigh, back, and abdomen. It consists most often of circular patches, brownish red like the lean of ham, dry, shiny, and inelastic; the patches run together, and form larger areas of irregular shape, but mostly with a well-defined edge. It is not always easy to distinguish this from eczema intertrigo in the situations liable to this last eruption; and the two conditions probably co-exist sometimes. Less frequently the rash is papular, pustular, or bullous. Other lesions occurring in early infancy are stomatitis, ulcerations about the lips and angles of the mouth, rapidly forming cutaneous abscesses, and periostitis. With all this the nutrition of the child may be little affected, but sometimes wasting results, and the face acquires a withered and shrunken appearance like that of an old man. In this stage death may occur; but under treatment, or otherwise, all the symptoms may subside, and the child may show no indications of the taint for many years, when, often about the time of puberty, symptoms appear which are more or less comparable with those of the third stage of the acquired disease.

Later Symptoms.—These are: periostitis, with the formation of nodes; synovitis; scaly or lupoid skin eruptions, which are not very common; bilateral deafness coming on with noises in the ears, but without pain or discharge; disseminated choroiditis; iritis and keratitis. The last is common in inherited syphilis—not so in the acquired disease; it causes opacity of the cornea, which gradually increases till the cornea looks like ground glass. It is associated with ciliary congestion, and in late stages vessels may encroach upon the cornea, producing a "salmon patch." Its tendency is to recover. But in addition to these fresh lesions, inherited syphilis may be recognised by some permanent deformities, the result mostly of those changes which took place in infancy. Such persons present a broad forehead, with unusual prominence of the two halves of the frontal bone; the bridge of the nose is broad and sunken; around the mouth are numerous linear cicatrices radiating from the orifice as a centre; and the permanent teeth, as was first pointed out by Hutchinson, show features from which alone an absolute diagnosis of the condition may be made. It is only the upper central incisors that can be relied upon for this purpose, though other teeth may be similarly affected: they are short, narrower at the edge than near the gum, and the edge presents a single central cleft or notch. This notch is at first, soon after the eruption of the tooth, filled by a notched edge of exposed dentine, which soon breaks away. This change in the teeth must be distinguished from the simple transverse marking, which may result from the excessive use of mercury in infancy, causing stomatitis, and interfering with the proper development of the tooth-sacs. An interstitial hepatitis is another important result of congenital syphilis (*see* Cirrhosis of the Liver).

Treatment.—Mercury acts with remarkable rapidity in infantile syphilis. A grain of grey powder three times a day, or liq. hyd. perchlor. 20 or 30 minims, will quickly cure the rash, snuffles, or other symptoms, and improve the nutrition of the child, if it is defective. If these cause purging, unguentum hydrargyri (10 grains) may be rubbed into the palms and soles night and morning. Iodide of potassium is of less value, but may be given in doses of 2 or 3 grains three times daily. To mucous patches, or ulcerated skin diseases, calomel powder or mercurial ointments may be directly applied.

TUBERCULOSIS.

By tuberculosis is meant the formation in one or more organs of certain bodies called "*tubercles*"; and these are now known to depend upon the presence of a specific micro-organism—the *bacillus tuberculosis* of Koch.

A tubercle in its most typical form is a small firm nodule, which consists of one or more *foci*, having the following structure:—Externally *lymphoid* cells, within these *epithelioid* cells, and in the centre a *giant cell*, with several nuclei. Sometimes there is a delicate reticulum or stroma, and invariably the characteristic *bacilli* are present in one or other part, especially in the neighbourhood of the giant cell. Sometimes the giant cells are absent, sometimes also the epithelioid cells, so that the tubercle may consist only of lymphoid cells.

This typical form is shown by the structures known as *grey* or *miliary* tubercles, which have a pearly grey or sometimes yellowish-grey colour, measure from one to two millimetres in diameter, and are well seen in the lungs, liver, and kidneys.

As the tubercle enlarges, it undergoes a process of necrosis, either from deficient vascular supply, for no vessel penetrates within the tubercle, or as a result of some chemical substance secreted by the bacilli. It becomes opaque yellow and cheesy in the centre, and the caseous matter, examined under the microscope, shows shrunken leucocytes, fat granules, and *débris*. The caseous centre enlarges, while at its periphery the tubercle may be invading more and more of the organ in which it is situated, the new tubercle becoming cheesy in its turn. In this condition it is known as a *yellow* or *cheesy* tubercle. In the solid organs, large spherical caseous masses are formed, as may be seen in the brain, and to a less extent in the spleen. If the tubercle formed originally on a surface communicating with a duct, the centre of the cheesy tubercle may break down, and be discharged, so as to leave a kind of ulcer, which enlarges by a continuance of the same process: this may be seen in the lung, the intestine, and the pelvis of the kidney. Some caseous masses ultimately undergo *calcification* (largely by calcium phosphate), by which the tubercle bacilli are destroyed, and the lesion ceases to be infective.

Another way in which tubercle terminates is by a *fibroid change*; chronic inflammation and induration of the surrounding tissue takes place, and the tubercle itself shrinks into a fibrous nodule. This is more common on the surface of the pleura and peritoneum, but also takes place in the lungs.

Tubercle Bacilli.—These are minute rods, straight, or very slightly curved, measuring ·003 mm. or 3 μ in length—that is, about half as much as the diameter of a red blood-corpuscle—and their breadth is about one-sixth of their length. They have rounded extremities, and present two or more bright spots, often one at each end, which were at first regarded as spores. Like other micro-organisms they can be stained by special reagents, and a method of detecting them by this means in the sputa is described hereafter (*see* Diagnosis of Phthisis).

Experiment has clearly shown that the introduction of tubercle-bacilli into the tissues will produce tubercles; and this must be by some special irritant properties of the bacillus. The bacillus may enter from without through the mucous passages, of which the respiratory gives us the preponderating number of instances; thus, tuberculosis of the lung follows the lodgment of the bacillus in the bronchioles or lung tissue. It may be introduced into the alimentary canal, either by milk or by meat supplied from animals, which are the subject of tubercle. With comparative rarity the bacillus may enter through a wound. In all cases we must suppose a special disposition on the part of the individual (*e.g.*, heredity), or of the tissue first affected (*e.g.*, depressed vitality from inflammation), which allows the tubercle to establish itself and thrive. This part of the subject is further considered in the chapter on Phthisis.

When it has once gained a hold on any one part, it may remain more or less localised, or it may, after a long time, spread throughout the whole of the body. The result, in the latter case, is the development of miliary tubercles in a number of organs at once—*e.g.*, the lungs, liver, spleen, kidneys, and cerebral meninges.

What here follows is an account of the symptoms which usually accompany this generalisation of tubercle. The symptoms of its local deposition in the various organs are described in other chapters (*see* Tubercular Meningitis, Phthisis, Tubercular Peritonitis, Tubercle of the Kidney, &c. &c.).

he choroid of the eye, the heart, thyroid body, marrow of
and peritoneum are involved. But the organs in the first
are not all diseased in every case; sometimes one, some
nother being spared. In any case, the only organs which
ise to definite local symptoms are the lungs and the
es of the brain; the other symptoms are toxic in kind,
h as are produced by other infectious processes. When
l symptoms are present early in the case, they generally
lmost entirely the pulmonary condition, and the case is
d as one of tubercular meningitis. This disease will be
ely described, and it will be noted that after death in
ses a tuberculosis of the lungs and other organs may be
without any clinical warning that they were implicated.
se of acute tuberculosis is fatal without any cerebral
ns and without tubercle of the meninges, then the tuber-
. be certainly found in the lungs, and of these cases in
e symptoms are obviously pulmonary, while in others they
re general. Thus cases of miliary tuberculosis may be
into three groups:—(1) Those with predominant cerebral
ns (tubercular meningitis); (2) those with predominant
ary symptoms (pulmonary tuberculosis); (3) those with
symptoms of infectious disease, in which also the lungs
erally involved, and which may also present even some
symptoms. It is of the last two forms, in which pul-
tuberculosis is a part of general tuberculosis, that the
section will deal.

logy.—A general infection with tubercle occurs as a com-
n of local tubercular diseases in various parts of the body;
ance, the lungs, the bones, the lymphatic glands, or the

after measles or whooping-cough, and very rarely after typhoid fever.

Anatomy.—It is in miliary tuberculosis that one finds the most typical examples of tubercle. Through the lungs the tubercles are, as a rule, uniformly scattered more or less thickly; occasionally only they may show a greater preference for the apices. Every form of tubercle may be seen—the grey, hard granulations, or the larger caseating tubercles, and sometimes these may be breaking down in the centre, forming minute cavities. Definite patches of pneumonic consolidation occur, but are not common. Some inflammation of the bronchi, especially the smallest, is always present.

Tubercles are sometimes found on the pleuræ, and pleurisy is often the result. In cases grafted on a former phthisis, consolidation and cavities will also be present. In the other organs mentioned tubercles are also found, of different ages in different cases. Tubercle bacilli have been found in the blood both during life and after death.

Symptoms.—These are at first and often throughout very obscure. The patient complains of weakness, inability to do his work, loss of flesh, anorexia, with nausea, or sickness, and headache. The bowels may be constipated or occasionally loose for a few days. Pyrexia is present, of a somewhat irregular type, generally high in the evening and much lower in the morning; thus the evening temperature may range from 100° to 103°, the morning temperature from 98° to 100°. Sometimes the urine contains a trace of albumen. Physical examination in other parts may reveal nothing, and such cases are often for a time thought to be enteric fever, though the absence of spots, of distension of the abdomen, and of characteristic diarrhœa makes such a diagnosis uncertain. If after a time some rhonchi or râles should be heard in the chest, the resemblance to typhoid fever is none the less; but there is often more pallor and lividity of the face, and more rapidity of breathing in proportion to the pulse and the physical signs of the chest than is common in enteric fever. The patient gets more emaciated, the pulse is rapid and feeble, the tongue dry, food is taken badly, and prostration becomes marked. Pulmonary symptoms may then become more prominent, such as cough and scanty mucous expectoration; and on auscultation more or less extensively diffused râles may be heard, or some cerebral symptoms may manifest themselves, such as strabismus, unilateral ptosis, weakness of one leg, twitching in one or other limb, or general convulsions. The prostration increases, there may be coma, and death is the result. The duration is from four to ten or twelve weeks.

In other cases the pulmonary symptoms are from the first more marked. Here also the loss of strength and emaciation are pronounced, but quite early there occur cough, dyspnœa, scanty mucous expectoration, tinged it may be with blood, and sometimes pain in the side. The physical signs are at first suggestive of bronchitis. Resonance is but little affected, there may be a slight impairment at one apex, or, on the other hand, some increase of resonance over the whole chest. With the stethoscope one hears sibilant and sonorous rhonchi, fine and small râles, of which many are consonating. Only occasionally one gets scattered patches of high-pitched breathing or obscure dulness. If the condition is secondary to an old phthisis the signs of this will of course be observed at the same time. When these conditions are well marked the patient presents a high degree of cyanosis—the face, lips, nose, ears, and cheeks being livid and the fingers shrunken and blue. The temperature has the characteristics already noted. Sometimes the *typus inversus* is present, the morning temperature being high and the evening low.

FIG. 9.

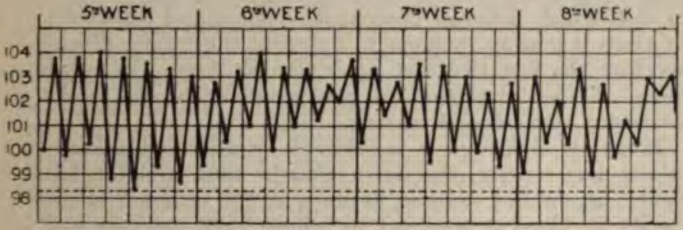

Chart of a Case of General Tuberculosis, fatal at the beginning of the Tenth Week.

Death at length takes place, after from three to eight or ten weeks, with increasing dyspnœa, lividity, prostration, or drowsiness. The ophthalmoscope should not be forgotten, as choroidal tubercles may be seen in some cases, and optic neuritis where the meninges are sufficiently implicated.

Diagnosis.—The insidious beginning, and the comparatively rapid prostration with a febrile illness, easily lead one to confound this disease with enteric fever, and the symptoms of bronchitis, as long as they are moderate, rather increase the difficulty. The points in favour of enteric fever would be rose spots, typical diarrhœa, and distended abdomen; those in favour of miliary tuberculosis are rapid emaciation from the first, the mixed pallor and cyanosis of the face, as contrasted with the pink flush and white face of enteric fever, and the rapidity of breathing out of proportion to the other signs of illness. The occurrence of any cerebral symptom should at once give the clue to the nature of the disease, and in any doubtful case the fundus of the eye should be examined for tubercles. These, however, are present in only a minority of cases, and optic neuritis is less likely to give assistance, since it is not generally seen before the onset of the cerebral symptoms themselves, and, moreover, occasionally occurs in enteric fever. The pronounced pulmonary cases are more likely to be confounded with bronchitis or broncho-pneumonia. The high fever and the rapid emaciation, combined with bronchitic signs and marked cyanosis, should help to distinguish acute tuberculosis. Cases of broncho-pneumonia present greater difficulties; the physical signs, the remittent pyrexia, and some cyanosis are common to both. Generally, the shorter duration, or the presence of rather decided areas of consolidation, will point to broncho-pneumonia; but broncho-pneumonia may last long enough to be mistaken for tuberculosis. When a child has lung complications after whooping-cough it is often very difficult to distinguish between broncho-pneumonia and tuberculosis; in this case it is more common to diagnose the former disease or to overlook the fact that the latter may be there. It may be suspected if the symptoms are prolonged for several weeks with increasing cyanosis and wasting.

Prognosis —Though it is believed by some that miliary tuberculosis may recover, the cases must be rare, and the tendency would be, in the absence of unequivocal evidence of tubercle (bacilli in blood or sputum), to believe that the case had been either enteric fever or broncho-pneumonia.

Treatment.—This must be almost purely symptomatic and supporting. Abundant fluid nourishment and small doses of stimulants should be given; opiates may be administered to relieve pain or distressing cough, and ammonia as an expectorant.

LEPROSY.

(*Lepra. Elephantiasis Græcorum.*)

A chronic infectious disease caused by the *Bacillus Lepræ* (Hansen), and characterised by nodular lesions of the skin, mucous membranes, and nerve-trunks. It is not common in Europe, except in Norway, and is, as a rule, only seen in England in the case of patients who have lived in the East or West Indies. But it is found in some parts of South Europe, and, amongst other places, in India, Burmah, Siam, China, Japan, the North-East of Africa, the Cape of Good Hope, West Indies, Mexico, Central America and parts of South America, and many Islands of the Pacific. It is not at all clear what determines its introduction into the body. It is not peculiar to one kind of climate or soil, nor can it be shown to result from any special kind of diet, though an exclusive fish diet has been alleged as a cause. Heredity seems to have but little influence. Direct transmission has only occasionally been proved, and nurses have lived in leper institutions for years with impunity; but it has been successfully inoculated, and it is in all probability contagious. It occurs in young people of both sexes.

Symptoms and Course.—It often begins with some general indisposition or malaise, with slight fever; and the lesion of the skin first shows itself as red or brownish-red spots on the limbs or trunk (*Lepra maculosa*). They may be from half an inch to three or four inches in diameter, are round or irregular in shape, and slightly swollen. Sometimes they form rings by clearing up of the centre of a field. With the subsidence of the pyrexia they may also fade, but are apt to leave pigmented stains, or sometimes white spots behind them; and from time to time fresh illnesses and fresh outbreaks of spots occur.

In the form known as *Lepra tuberculosa* or *L. nodosa* there occur, simultaneously with the spots, or after an interval, hard elevations of the skin, from the size of a pea to that of a hazelnut or larger. They may persist for a long time, or may ultimately disappear, leaving pigment spots behind; or they may soften, break through the skin, and leave indolent ulcers, having weak granulations, and discharging a scanty thin pus. The nodules appear mostly on the face, on the dorsal surfaces of the hands and feet, and on other parts of the limbs. On the face they are apt to produce great deformity by the enormous thickening of the eyebrows, the nose, cheeks, and lobes of the ears; and the very characteristic appearance thus produced is described as *leontiasis*, from its resemblance to the face of a lion. The tubercles also develop in the mucous membranes of the mouth, the gums, the palate, the larynx, or the nose; and the voice may be rough and hoarse, or high-pitched and feeble in consequence. The ulcers may eat deeply into the parts beneath, so as to erode tendons and bones, and open the joints.

In *Lepra anæsthetica* nervous symptoms predominate, but they often coexist with the conditions above described. They consist partly of tingling, partly of numbness and actual anæsthesia, as proved by the inability to feel the prick of a needle-point. This is found in various isolated patches on the limbs and trunk; and over these patches the skin is either paler than normal or more pigmented, the hairs are small and wanting in pigment, and the skin generally is smooth and glistening. In this form, also, deep ulcerations take place over the joints of the fingers and toes, and the phalanges, or carpal or tarsal bones, may ultimately be shed;

[illegible] often being spared, and the [illegible] with remarkable completeness [illegible]

[illegible] the disease has a hopeless [illegible] loathsome object to look at, but [illegible] of appetite, or otherwise inter- [illegible] vital functions. Death takes [illegible] years, from tuberculosis of the [illegible] current affection; and occasionally [illegible] gangrene, or pyaemia, the more [illegible]

[illegible] the skin beneath the epidermis, [illegible] subcutaneous tissue. The nodules [illegible] (*lepra cells*), varying in size from [illegible] four times as large, and at first [illegible] The cells contain the bacilli [illegible] resemble tubercle bacilli, but [illegible] in always being straight. In [illegible] the seat of neuritis. They are [illegible] times their normal size, from [illegible] which finally the axis-cylinders [illegible] Nodular infiltrations also form, [illegible] the skin in the case of superficially [illegible] has been found not [illegible] skin and of the nerves, but also in [illegible] kidneys, and rarely in the lungs.

Treatment. [illegible] incurable. The most [illegible] the patient to a locality in which [illegible] give good food and cod-liver oil, and [illegible] as may be present. Chaulmoogra oil, [illegible] increasing from 20 minims to [illegible] to do some good.

PYÆMIA.

[illegible] by febrile reaction of an inter- [illegible] with the formation of abscesses in various parts or [illegible] which are secondary, in the great majority of cases, [illegible] existence of an open wound, accidental or opera- [illegible] of pus in some part of the body. Hence it [illegible] surgical disease, and was in former times [illegible] surgical wards of a hospital, until the almost [illegible] antiseptic methods of the treatment of wounds [illegible] But it has a medical interest as well, since it is essentially a general disease; it may arise from lesions which come frequently under the notice of the physician, such as ulcerations of the mucous surfaces which are not amenable to antiseptic treatment; and it occurs exceptionally without any preceding lesion, so far as can be ascertained by the most careful examination, whether during life or after death (so-called *idiopathic pyæmia*).

With this exception, the association of the disease with the primary lesion is quite obvious in all cases; and the name pyæmia (pus in the blood) arose from the idea that the pus was actually transferred from the original lesion to the seat of the secondary abscesses along veins in which no protective coagulum had been formed. It is now, however, clear that the disease is due to a double process—(1) the absorption of pyogenic organisms, and (2) the impaction of numerous emboli in different parts of the body. The organism is in most cases the *streptococcus pyogenes*, less commonly a *staphylococcus*. The emboli are fragments of thrombus, pus, or *débris*, which contain micro-organisms, and which, being arrested in the minute vessels of a part, not only cause *infarction*, in the same way as other non-septic emboli (*see* Embolism), but also set up inflammation and suppuration. The thrombus is frequently provided by the coagulation of blood in the veins in connection with the wound. Embolism is most common in the bases of the lungs, where the wedge-shaped or conical areas of lobular pneumonia and suppuration are the most characteristic feature of pyæmia. But abscesses occur in other parts of the body, such as the liver, spleen, and kidneys, which are not within the pulmonary circulation. Cocci are found in the secondary lesions, both in the capillaries and in the tissues; and they are seen, but not constantly, in the blood. The occurrence of abscesses in the range of the systemic circulation is not very easily explained. The embolic particles are not likely to pass through the capillaries of the lung, and it must be supposed that cocci which can so pass may become aggregated together to form emboli in the liver, spleen, and kidneys; or that the organisms are deposited in the endothelium of vessels and there grow, or perforate into the tissues and form abscesses.

Secondary abscesses, indeed, may occur in nearly every part of the body, but particular organs are associated together in ways that are partly, but not altogether, explained by the course of the blood-stream. (1) In the more common *acute fatal pyæmia*, the abscesses are nearly always found in the lung, and perhaps there alone. They are often associated with acute pleurisy, which may be serous or purulent; this is mostly referable to the proximity of the abscesses to the pleural membrane. Pericarditis and peritonitis also occur. (2) Another kind of case begins with acute necrosis of a bône; and the secondary abscesses form especially in the cardiac muscle and in the kidneys. (3) In portal pyæmia the primary lesion is mostly some form of ulceration of the intestine, and secondary abscesses form in the liver, with or without a suppurative pylephlebitis. (4) A fourth variety is infective endocarditis, which was called by Wilks *arterial pyæmia;* and in which micro-organisms and *débris* of thrombus are conveyed in the blood-stream to distant organs, and may produce suppurating infarcts, especially in the brain, kidneys, and spleen. This closely resembles ordinary pyæmia; indeed, it may have typical pyæmic symptoms, and form part of pyæmia itself.

In *chronic pyæmia* the viscera are mostly spared, abscesses form in the subcutaneous tissues, and the joints inflame or suppurate. This is not uncommon in puerperal cases. Watson Cheyne does not regard this as a true pyæmia, since the abscesses do not appear to depend on embolic processes. A similar objection would hold with regard to gonorrhœal synovitis and scarlatinal rheumatism being regarded as pyæmic in origin.

Ætiology.—Apart from accidental and operative wounds, the lesions which lay the body open to pyæmic infection are typhoid or dysenteric ulceration of the intestine, ulceration of the vermiform appendix, fistula, gonorrhœa, prostatic septic thrombosis, otitis media, and post-partum exposure of the uterine surface. Intemperance, such general conditions as Bright's disease, and acute fevers, are believed to predispose to the occurrence of pyæmia. The fact that so-called idiopathic cases occur is paralleled in other forms of infective disease, such as infective endocarditis and cerebro-spinal fever, where the mode of entry of the organisms is not always apparent.

Symptoms.—The disease often begins suddenly with a prolonged rigor, followed by profuse sweating and collapse, the temperature rises, and fever continues to be interrupted by fresh rigors daily, or two or three in the day, but often without any regularity. There are anorexia, thirst, and dry tongue; anxiety, prostration, rapid breathing, and loss of flesh. The face is usually sallow, or even distinctly jaundiced, and the urine may contain some bile-pigment. Sickness is not infrequent, and diarrhœa may be present. The rigors may cease after five or six days; but fever of an intermittent or remittent type continues; occasionally there are [illegible] erythematous patches in various parts of the body. As already stated, the local lesions vary, and the symptoms will [illegible] accordingly. When the lungs—as is common—are the [illegible] abscesses, the breathing is rapid, with supple[illegible] in front, deficient entry of air at the bases, [illegible] crackling râles; or there are dulness, tubular [illegible] signs of pulmonary consolidation or pleural [illegible] pericarditis will be shown by its character[illegible]. The duration of these cases is often quite short—

from six to ten days; a typhoid condition ensues, with prostration, stupor, delirium, dry brown tongue, quick feeble pulse, and death.

In the chronic cases, where the viscera are spared, and the abscesses form in the joints, the latter become swollen, tender, and hot; tender points appear on the surface of the limbs or body, and beneath them abscesses rapidly form, with thin, unhealthy pus and imperfectly developed limiting walls. Fresh abscesses occur from time to time for several weeks or months, and the patient may ultimately recover, sometimes with ankylosis of joints; or death may take place from exhaustion. The symptoms in other cases may be modified by the special localisation of the secondary lesions. In pyæmia secondary to otitis the lungs are implicated, or there may be pleurisy or empyema with the lung tissue nearly free. If meningitis occurs the cerebral symptoms will largely mask the others.

Morbid Anatomy.—In acute cases of pyæmia, in addition to the pulmonary abscesses and the inflammation of the serous membranes, there are found dark fluid blood, soft organs, and ecchymoses under the serous membranes.

Diagnosis.—The occurrence of rigors and profuse sweatings in the course of the treatment of a wound, followed by collapse and a typhoid condition, while the wound takes on an unhealthy appearance, is characteristic of pyæmia. Where the same symptoms occur without any external wound the same diagnosis may be obvious, and search will have to be made after the primary lesion, such as otitis, with discharge from the ear, disease of the nose, intestinal ulceration, or abdominal suppuration. Sometimes the rigors take place with such regularity as to resemble *ague;* the resistance to the influence of quinine and other points in the history will usually serve to distinguish the cases. *Infective endocarditis* may be generally distinguished by the presence of a murmur; but endocarditis of the pulmonary valves may be actually caused by pyæmia secondary to suppuration, as from wounds or other sources.

In the late stages pyæmia may closely resemble *enteric fever;* especially if there is no discoverable lesion to suggest it: a history of rigors would, however, be opposed to enteric fever. Lastly, joint pains, like those of *rheumatism*, within a few weeks of confinement or miscarriage should always excite a suspicion of pyæmia. In this disease the inflammation persists in each joint as it is involved; whereas, in rheumatism, the pains fly from joint to joint, and may return again in those first affected.

Treatment.—This is almost hopeless in the visceral forms; but less unpromising in those with synovitis and cutaneous abscesses. In all cases nourishment and stimulants must be given freely, and quinine (5 grains), or sodium sulpho-carbolate (10 grains), every four hours. If the primary lesion can be reached, it should be dealt with surgically, so as to get free drainage and antisepsis; and secondary abscesses should be opened where accessible.

Sapræmia and Septicæmia.

Two other forms of infective disease arise in connection with wounds and operations; they are due to the absorption of the products of bacteria or of the bacteria themselves, but are independent of the secondary abscesses, the presence of which constitutes pyæmia. Their symptoms are practically identical; and in either case they vary in intensity, presumably in proportion to the amount of poison in operation, consisting mainly of fever, with high temperature, prostration, quick feeble pulse, vomiting and diarrhœa, dry tongue, delirium, stupor, and the usual accompaniments of the typhoid state. The two forms are called respectively sapræmia and septicæmia. In ***sapræmia*** the symptoms are due to the circulation of poisonous substances (toxins or ptomaines), which result from the action of putrefactive bacteria upon necrosed tissues, without the penetration of the bacteria into the circulation. The poisons are eliminated with the urine, and their further production is at once checked by proper treatment (*e.g.*, antisepsis) of the wound or source of infection. In ***septicæmia*** the micro-organisms enter and multiply in the blood, and thus the continued production of poisons is only to a limited extent affected by local treatment.

These conditions, like pyæmia, must be treated by local antisepsis, and stimulating and supporting measures internally. For further details the reader is referred to works on Surgery.

TETANUS.

This disease, of which the name is derived from τείνω, I stretch, consists of a tonic contraction of most of the muscles of the body, with paroxysms of increased contraction from time to time.

Ætiology.—It occurs in quite young infants (***tetanus neonatorum***), and after that age, at all periods of life from five years upwards. It is more common in hot countries, and the dark-skinned races seem especially liable to it. A very frequent antecedent is injury (***traumatic tetanus***). This may be of any kind, from a mere scratch with a pin or nail, to the most serious compound fracture or lacerated wound; even blows and injuries which do not break the skin have been said to cause it. In newborn infants the division of the umbilical cord supplies the traumatic lesion; not infrequently, especially in tropical climates, the disease follows upon abortion or labour at full term. Cold and damp have been held accountable for some cases, but both these instances of *rheumatic tetanus*, and those in which no cause can be discovered (*idiopathic tetanus*) can only now be regarded as cases in which the point of entry of the specific virus has been overlooked. There is evidence of the disease having sometimes taken on an epidemic character.

Symptoms.—Within a few days of the occurrence of the injury in obviously traumatic cases, and without any warning in others, the patient feels stiffness at the back of the neck, and the same in the jaws, so that he is unable to open his mouth wide, or to masticate properly. He may continue like this for a day or two, or may rapidly pass on to the next stage, in which there is rigidity of the muscles of the trunk and to a less extent of those of the extremities. The back becomes rigid, and is slightly arched, with the concavity backwards (*opisthotonus*); the muscles of the trunk and abdomen become quite hard from constant contraction; the movements of the chest are limited from the same cause; the legs are generally rigid, but the arms are only a little stiff about the shoulders and elbows, and the fingers may be moved freely. By this time the jaw is generally firmly fixed by contraction of the masseters, and the teeth cannot be separated for more than a quarter of an inch (*trismus*, or lock-jaw, by which last name the disease itself is popularly known); the angles of the mouth are drawn outwards, and the lips are slightly separated; the eyebrows are drawn up by the frontal muscles, and together by the corrugators, so that the facial expression is that of a painful grin, known as the *risus sardonicus*. When this stage has been reached, the so-called "spasms" or paroxysms of increased and even violent muscular action begin. These consist of sudden contractions involving the whole of the muscles hitherto in tonic rigidity. The teeth are clenched more violently, the *risus* becomes more marked, the head is thrown back, and the back arched more strongly, the chest is fixed, and the respiratory process is checked; a groan may escape from the patient, either elicited by pain or the result of expiratory spasm. The paroxysm is often of momentary duration, scarcely to be counted in seconds, and the patient relapses into his former condition of tonic contraction; or it may last several seconds, with imminent danger to life, from the hindrance of respiration. It is always intensely painful; it is brought on by external impulses, by touching the patient, jerking his bed, by passing a catheter, or giving a subcutaneous injection. The paroxysms occur at first at intervals of half an hour, an hour, or more, but as the disease progresses unfavourably, they become more violent, and occur at shorter and shorter intervals. Between the spasms there is still some pain from tonic contraction, respiration is not entirely free, and the voice is feeble. The reflexes are increased. The pulse is small and quick, and becomes quicker during the paroxysms. The temperature generally at first remains normal, and may continue so to the end, though it sometimes rises a little before death; sometimes the temperature is constantly above normal; in other cases an extraordinary rise to 107° or 108° takes place just before death, and it has been observed to continue rising even after death to 110°. The urine is often retained, so as to require the use of the catheter. Sensation is generally unaffected, and the cerebral functions are mostly quite normal until near the end, when delirium may occur. In a great number of cases the disease progresses to a fatal termination in from one to twelve days: the paroxysms become more violent and frequent; and death takes place from exhaustion, or from spasm of the glottis, or from fixation of the respiratory muscles; pneumonia or bronchitis may add their influence against the patient. In a few cases life is prolonged to the third or fourth week. On the other hand, recovery may take place: the spasms, having reached their height, gradually become less frequent; the constant rigidity of the muscles subsides, and the patient is convalescent in from three to six or eight weeks.

Varieties.—Occasionally a case runs its whole course to a fatal termination without any paroxysms, in addition to the general rigidity. Very rarely paroxysms occur without the continuous spasm. Rose describes a "hydrophobic tetanus": this arises from injuries to the head, involving the distribution of the fifth nerve. The peculiarities of this variety are that the facial nerve is paralysed, and that spasms of the throat occur resembling those of hydrophobia. The terms *acute* and *chronic* are often used in reference to tetanus; but there is no essential difference between the cases of short, and those of long, duration.

Morbid Anatomy.—Many cases present after death no pathological lesions whatever. The organs most commonly affected are the lungs, which may be the seat of pneumonia, bronchitis, œdema, or hæmorrhages. The central nervous system, as a rule, looks perfectly normal to the naked eye. Microscopical examination also often fails to reveal anything, but sometimes shows minute changes which, however, do not in any way explain the clinical conditions, and are partly, no doubt, the result of the disturbance of the circulation during the paroxysms. The muscles of the trunk, especially the abdominal muscles, are sometimes ruptured, or the seat of hæmorrhages. In traumatic cases the wound may be in every possible condition—in a healthy state of repair, or actually cicatrised, or suppurating, or sloughing.

Pathology.—The specific cause of tetanus is a bacillus, the bristle-bacillus of Nicolaier, which appears to exist in different forms of earth or garden-mould, since it is developed in animals, together with symptoms of tetanus, when such earths are inoculated under the skin. Presumably in ordinary traumatic tetanus the bacillus is introduced at the time of the injury with the dirt, which is so often associated with accidents. The bacillus multiplies chiefly in the neighbourhood of the wound, and produces poisons which circulate in the blood and act upon the spinal cord. Tetanus has also been transmitted from man to animals by the inoculation of materials from the wound, and by the injection of urine, which contains the specific poison.

Diagnosis.—Tetanus may have to be distinguished from strychnia-poisoning, hydrophobia, spinal meningitis, tetany, muscular rheumatism, and hysteria. In ***strychnia-poisoning*** the extremities are involved to a much greater extent than in tetanus, and the paroxysms are excited by external stimuli; but in the intervals the muscles are relaxed. The symptoms develop very rapidly, but do not begin with trismus. In ***hydrophobia*** there is no continuous rigidity; the spasms involve the respiratory muscles, and are excited by the attempt to drink, or the sight of fluids. Mental agitation or even maniacal excitement is generally present. In ***spinal meningitis***, again, trismus is not an early symptom; nor is there constant rigidity; muscular spasms are excited by attempts to move, and the temperature is high from the first. The early occurrence of cerebral symptoms would be opposed to tetanus. The peculiar distribution of the spasm in ***tetany*** makes it easy to distinguish it from tetanus. ***Muscular rheumatism*** may cause stiffness of the back of the neck, which might, under certain circumstances, cause alarm; but trismus is never present. In severe forms of ***hysteria*** opisthotonus is often a prominent feature, but it occurs as part of a series of convulsive movements, which cannot be mistaken. Trismus may occur in hysterical patients, but is not accompanied by rigidity of the cervical muscles, and is variable in intensity. Other indications of hysteria, as, for instance, a preceding convulsive fit, may be present.

Prognosis.—Amongst traumatic cases, about 90 per cent. die; and of other cases about 50 per cent. Tetanus is very fatal after pregnancy and abortion. On the whole, recovery is less common in cases due to severe and extensive injuries than in those due to slight injuries; and it is less frequent when the symptoms develop rapidly after the injury, or run a rapid course to a severe stage, than when they come on in every way slowly.

Treatment.—The patient should be kept at rest, and is best placed in a darkened and perfectly quiet room, so as to avoid all impressions of sight and sound. Nourishment should be given freely, in fluid form; but the closure of the jaws may necessitate its being given by a nasal tube, or by the rectum. If there is already a deficiency in the teeth, a tube may be passed into the mouth; but the extraction of a tooth for this purpose is of doubtful expediency. It has been suggested that once or twice daily chloroform may be administered, so as to relax the jaws, and enable food to be given. In traumatic cases, the wound should be treated thoroughly with antiseptics, so as to remove or destroy as far as possible the bacilli from which the poisonous materials are proceeding. But this is not in itself sufficient, as the appearance of the symptoms is proof that the poison is already circulating.

In tetanus, as in diphtheria, the treatment by an *antitoxin* has been attempted. Indeed, Tizzoni and Cattani had already used this method long before the serum treatment of diphtheria: and preparation of the tetanus antitoxin is very similar. Unfortunately the success of this proceeding has not been great: the cases which have recovered under it being chiefly those, the less acute cases, which would have got well on other plans of treatment.

Many drugs belonging to the classes of anodynes and spinal sedatives have been employed in the treatment of tetanus; but they can only be regarded as palliative. They are practically powerless against the most acute and severe forms; in less violent cases some of them are of benefit by relieving pain or diminishing spasm; but this last effect will only be gained by the employment of full doses, which, in the case of some drugs, must be carefully watched to prevent the production of other toxic effects. Chloral, bromide of potassium, and Calabar bean seem to have been most successful. Chloral may be given in doses of 30 or 40 grains every four or six hours, and even drachm doses have been given without harm; and bromide of potassium may be given in drachm doses frequently; or these two drugs may be combined. The extract of Calabar bean may be given in 1-grain doses every two or three hours; or the sulphate of eserine may be injected subcutaneously in doses of $\frac{1}{6}$ to $\frac{1}{4}$ grain every three hours, until its toxic effect is shown in fibrillary twitching of the muscles, and diarrhœa. Morphia injections or opium may also be given once or twice daily to procure sleep; but the continuous use of opium is not so successful as that of the above-mentioned drugs. Belladonna and atropine, aconite, cannabis indica, nitrite of amyl, and curare have also been employed, but with even less encouraging results. In most cases it will be found expedient to give some stimulant with the food.

HYDROPHOBIA.

This is an infectious disease, which is invariably caught from animals suffering from an allied disorder named *rabies*. This occurs in the wolf, fox, cat, cow, and horse, but much more frequently in dogs; and it is the bite of a dog, which, as a rule, by means of the saliva, introduces the poison into the human blood. Rabies in animals has been described as occurring in two forms, but they are not essentially different. In the first, or *furious rabies*, the dog is at first low-spirited, timorous, and unwilling to move; he then becomes suspicious and irritable, with a strong tendency to bite, and often with a peculiar howl. He refuses his ordinary food, and will eat straw, earth, hair, clothes, bits of wood, &c. Paralysis supervenes, the lower jaw drooping, the limbs failing, so that the animal can no longer stand, and finally death takes place. In *dumb rabies* there is no maniacal stage; the paralytic symptoms appear early, and are soon fatal. In neither case is there the fear of water which gives the name to the human complaint.

Ætiology.—It appears that in only about half the cases of bite by mad dogs does hydrophobia afterwards develop. This is more likely to be the case if the bite is on an exposed part, such as the face or hand. A portion of clothes driven in by the tooth may protect from infection. If an abraded surface, or even perhaps a mucous membrane, is licked by a mad dog, infection may occur. The disease is more frequent in men than women, from their more frequent association with dogs; it may occur at all ages.

Symptoms.—After the inoculation there is a period of *incubation* which is of remarkable length: in the majority of cases it is from two to nine weeks, and in some cases it is several months. During this time there may be absolutely no symptom. The first definite sign is often an uneasy sensation or pain in the scar of the wound. This pain may be very severe, and the scar may be slightly reddened or tender. But these indications may be entirely absent, and then the first sign is a feeling of malaise or depression, restlessness, sleeplessness, irritability, failing appetite, with a sense of choking or an uneasy feeling about the throat. Then appear the spasms, which are so characteristic of the disease: they are excited by the attempt to drink, by the sight of water or the vessel containing it, or by the suggestion of those around that some fluid should be taken. Later on they are induced by almost any external impression—a breath of air, a flash of light, or a loud noise. The spasms involve the muscles of deglutition, but the most obvious are those affecting the muscles of respiration—a sudden deep inspiration, like a sob or sigh, is made, the shoulders are raised, the chest expanded, and the sterno-mastoids or platysmas contracted. If water is forced upon the patient, more voluntary efforts to reject it are made, and an aspect of fright or terror is assumed. After a time the convulsions extend to other muscles of the body, presenting a tetanoid character. The difficulty of deglutition is shown in another way, for the saliva is not swallowed, but is constantly being collected in white frothy pellets, and is expectorated in all directions, or in the faces of those around. With the increasing severity of these spasms, the patient becomes excitable, talkative, delirious, or wildly maniacal, with delusions and hallucinations. The temperature is raised, the face is flushed, all attempts to give food may be futile—at most a small quantity of milk or other nutriment may be gulped down in a moment of greater control. Emaciation is remarkably rapid in the small time the disease lasts, and exhaustion necessarily follows. Not infrequently, towards the end, the spasms cease entirely, and the patient may even take good quantities of food; but even if this is so, it does not avail to prevent the fatal end, which may be preceded by paralysis or coma.

The duration of the disease is from two to four days; ten days seem to be the longest period known. Death is almost invariable.

Anatomy.—Gowers has found microscopic changes in the nervous system, especially in the cortex of the brain, in the spinal cord, and most abundantly in the medulla oblongata. They consist of dilatation of vessels, collections of small cells round the vessels and in the tissues, clots in the vessels, and small hæmorrhages. Such changes, however, are not constant. Leucocytal infiltration has been also seen in the salivary glands and in the kidneys. The microbe of hydrophobia has not yet been identified.

Diagnosis.—This is not generally difficult, especially if the fact of infection is well authenticated. There is but little real resemblance to tetanus, in which the permanent rigidity of muscle and the absence of mental disturbance are distinctive. Hysteroid conditions may simulate hydrophobia, and may occur where the mind has been much directed to the possibility of hydrophobia coming on. Globus hystericus may be regarded by the patient as a "spasm" of the throat.

Treatment.—When the disease is once developed little can be done. No remedy is known with certainty to have any influence upon it. Relief may be given by morphia injections, or chloroform inhalations. Cases are reported to have been cured by curare; if it is used, a quarter or half grain should be injected every quarter of an hour till paralysis occurs, and repeated as the effect passes off. But, as already stated, the cessation of spasm is not the cure of the disease.

Prevention.—On the occurrence of a bite, a ligature should be at once placed above the bitten part (if it is a limb) and the wound may be sucked, so long as the mucous membrane is unbroken, and the mouth is frequently rinsed after ejecting the blood. The wound should then, as soon as possible, be cauterised with nitric acid, or nitrate of silver, or it should be excised.

In 1885, M. Pasteur first communicated to the Paris Académie des Sciences a method for preventing hydrophobia by inoculation of the virus of dog's rabies, modified by transmission through rabbits, and by subsequent exposure to air. A rabbit is trephined and inoculated under the dura mater from the spinal cord of a rabid dog; the rabbit becomes rabid after fifteen days' inculation. A second rabbit is inoculated from the first, a third from the second, and so on until the period of inculation, which grows shorter with successive inoculations, is reduced to the minimum of seven days. The spinal cords of these rabbits contain the virus in every part; but if a fragment be separated, and suspended in dry air, the virulence gradually diminishes, and disappears in a period of time, which varies with the size of the fragment and the temperature of the air. For the purposes of preventive inoculation a number of fragments of the virulent spinal cords of rabbits are kept in separate bottles of dry air, the date of their introduction being noted. A healthy dog is then injected subcutaneously with a certain quantity of one of these cords that has been dried sufficiently long to destroy the virus--say fifteen days; on the next day spinal cord that has been dried fourteen days is injected; on the next day spinal cord of thirteen days' drying; and so on, on successive days, until spinal cord is injected which has only been dried one day. The dog is then found to be incapable of contracting rabies. Pasteur then tried the same method on persons that had been bitten by rabid dogs and feared hydrophobia, and this with such apparent success that hundreds from all countries have placed themselves under his hands for inoculation. Modifications have been since made; in the *intensified* method, the inoculations follow upon one another with much greater rapidity. There is no doubt that hydrophobia has been largely prevented by this treatment, the percentage of cases in which disease has developed after the inoculation being much less than what was regarded as the average.

An antirabic serum has also been prepared.

GLANDERS.

(*Equinia*, *Malleus*, *Farcy*.)

Glanders is a disease which affects chiefly horses, mules, and asses, though sometimes other domestic animals, and is occasionally transmitted accidentally to men. Grooms, stablemen, and others in charge of horses are most liable to contract the disease, which, in its acute forms, is a febrile disorder, characterised by special lesions of the nasal and respiratory mucous membranes, by the formation of subcutaneous nodes and the implication of the lymphatic vessels and glands, and by a cutaneous eruption. It also occurs in a chronic form. The term *Farcy* was given to cases in which the subcutaneous nodules (farcy-buds) with the lymphatic lesion were the prominent features; but it is not desirable to have two names for one disease, and glanders is now the appellation generally adopted.

The disease is mostly transmitted to man by accidental inoculation of wounds, cuts, or abrasions, either in grooming a glandered animal, or in skinning one dead of the disease; or a horse may bite its groom, and convey the disease by means of its saliva, or may sneeze and discharge some nasal mucus into the eye, nose, or mouth of any one standing near. It is stated that it may be conveyed by eating the raw flesh of a glandered animal, and that it has been caught in this way in menageries. It may also be communicated from man to man.

The bacillus of glanders (*B. mallei*) is found in the nodules; it is about the size of the tubercle bacillus. It has been artificially cultivated, and inoculations have been made, leading to lymphatic inflammation and general infection, with the formation of nodules and ulcers on the nasal septum, and nodules in the lungs, in which again the bacillus has been found.

Acute Glanders.—The disease begins with malaise, headache, lassitude, loss of appetite, and pains in the joints and limbs. For a time there is often a resemblance to rheumatic fever or enteric fever, or there may be pain in the side and dyspnœa. If a wound or scratch has been infected directly, it becomes inflamed, tense, painful, and the skin around has the appearance of erysipelas. The sore ulcerates, and discharges a sanious fluid, and the lymphatics in the neighbourhood may become enlarged. The more characteristic features of the disease may not appear for a week or more after its commencement, though sometimes earlier. The *eruption* consists of small red papules, upon which vesicles appear; these soon form bullæ, or pustules of different sizes, up to half or three-quarters of an inch in diameter, hemispherical, flat, or depressed in the centre, with serous, purulent, or blood-stained contents. The base of the pustule is inflamed, and infiltrated for some distance round. After a time, the discharge escapes, and an ulcer covered with scab or slough remains. The *nodes* which form under the skin are at first hard and painful, and generally suppurate; they also frequently occur in the muscles. The lymphatic glands are not always inflamed. The implication of the mucous membranes is shown by a discharge from the nose, which is at first a thin mucus, but afterwards becomes thick, viscid, purulent, fœtid, and often blood-stained. It is connected with the formation of tubercle-like nodules on the nasal mucous membrane, which caseate, ulcerate, and may perforate the septum nasi, or destroy the turbinate bones. Other mucous membranes may be affected—*e.g.*, the conjunctiva, and those of the frontal sinuses, the pharynx, the larynx, and the bronchi. In the lung are found deposits which caseate and suppurate, as well as patches of lobular hepatisation.

The progess of the case is generally downwards, with symptoms of a pyæmic or adynamic character. The temperature is high, but may oscillate; the pulse is quick; and the tongue dry and brown. Albumen appears in the urine, low delirium with tremors is succeeded by coma, the breathing becomes more rapid, and death finally ensues, generally in two or three weeks from the commencement.

Chronic Glanders.—Here the local lesions predominate. They consist of ulcers with thick and hard edges, or abscesses about the joints, or inflammatory swellings beneath the skin, or in the muscles. Or a pustular eruption may occur, but it develops more slowly than in the acute form. The nasal mucous membrane may also be involved, and in some cases emaciation occurs with hoarseness and pulmonary symptoms, such as cough and hæmoptysis. The average duration of the chronic cases is stated to be four months.

Pathology.—On *post-mortem* examination of acute glanders, changes characteristic of pyæmia are often found: increased fluidity of the blood, and abscesses of the lungs, the pyæmia being secondary to the local lesions.

The characteristic lesions of glanders are found in the mucous membranes, the skin, and the lungs. In the nasal mucous membrane, subepithelial nodules occur, from the size of millet seeds up to that of a pea, consisting of lymphoid corpuscles, or pus-corpuscles. In a later stage these have suppurated, and left ulcers with yellowish bases. Around these fresh nodules of infiltration have formed, which go through the same process. If recovery takes place, irregular puckered scars are left. In the lungs, similar nodes form, the centres breaking down into a caseous detritus. These are accompanied with patches of broncho-pneumonia, which may form abscesses. Similar nodes form in the intestinal mucous membrane, in the skin and subcutaneous tissue, and in the muscles.

Diagnosis.—In early stages the disease may be mistaken for rheumatism or typhoid fever, later for pyæmia. In chronic cases, syphilis, scrofula, and phthisis may be simulated. In veterinary surgery, the diagnosis is made by the injection into suspected animals of *Malleïn,* which consists of the chemical substances present in the artificial cultures of the glanders bacilli. A definite "reaction" with rise of temperature occurs in diseased animals, similar to that produced in man by Koch's *tuberculin* (*see* Treatment of Phthisis).

Prognosis is very unfavourable. Only a few recoveries from acute glanders are recorded; and only about half of the chronic cases get well.

The **Treatment** must be supporting and stimulating. Quinine should be given internally; the nasal lesion should be treated with antiseptic injections, such as creosote, carbolic acid, iodine or potassium permanganate lotions. Abscesses of the skin should be opened when ready. For chronic cases, carbolic acid, potassium iodide, arsenic, strychnia, and sodium benzoate have been recommended.

ANTHRAX.

This term, formerly the Latin equivalent of *carbuncle*, is now generally used to designate a disease which affects various animals, and is communicated from them to man. In animals it is known as *splenic fever;* in man it includes *charbon* of the French, and *malignant pustule* of English writers. Its distinguishing feature is the presence of a bacillus (*B. anthracis*), which can be found in the local lesions, the blood, viscera, and secretions. It consists of a motionless, short, homogeneous rod, straight or slightly curved, varying from $\frac{1}{2500}$th to $\frac{1}{1250}$th of an inch (10—20 μ) or more in length—that is, considerably larger than the diameter of a blood-corpuscle. The rods multiply by elongating and dividing, and also produce within themselves spores, which subsequently become free and reproduce the rods. The spores have great vitality, resisting considerable changes of temperature.

Among animals this disease can be conveyed by direct inoculation, probably the bites or stings of insects, or the bites of dogs that have eaten the flesh of animals dying of the disease. It is also transmitted indirectly by animals feeding in damp meadows or on moist soils, where the specific micro-organism contained in the dejecta of previously diseased animals may have been preserved in an active condition; and, according to Pasteur, the bacillus multiplying around buried carcasses may be carried to the surface by earth-worms, and distributed on the vegetation.

Infection in man occurs from the living animal, as in drovers, shepherds, and farmers; but much more often from the carcass. Thus, slaughterers, butchers, and those who have to do with the hides may be infected through a scratch or wound, and, rarely, it may be contracted by eating the flesh of diseased animals. Most frequently, however, in England it occurs amongst tanners and those who have to handle the skins and hides that come from abroad, and amongst those who deal with wool and hair from the same animals. Thus, wool-sorters, furriers, tanners, and others in like occupations may contract the disease either by direct inoculation through the broken skin, or by inhalation of dust or wool particles proceeding from the goods. In Bradford it has been known as *wool-sorters' disease*. Rarely it is transmitted from man to man by direct contact.

Symptoms.—Different varieties of the disease are described. These are local or external anthrax—*malignant pustule* proper; and internal anthrax, which includes a pulmonary and a gastro-intestinal form. Either of these two may be combined with the local variety.

Malignant Pustule.—Infection generally occurs through a scratch or abrasion on the face, neck, hands, or arms. After an *incubation* of a few days, or it may be only some hours, the spot itches or burns, and a small pimple appears, which vesicates, and the vesicle bursts and discharges a thin fluid. The base of the vesicle then forms a brownish or black eschar, and the skin around becomes red, swollen, and indurated, forming a prominence from one and a half to two inches or more in diameter. Around the central eschar there is often a ring of small vesicles containing serum, and the skin for some distance round may be œdematous, and the nearest lymphatic glands enlarged and tender. For three, four, or five days the patient may feel in his usual health and continue at work; he then becomes feverish, with prostration, delirium, sweating, or diarrhœa, and finally, in many cases, death occurs, preceded by collapse.

In *malignant anthrax œdema* no definite pustule forms, but an œdematous swelling, usually affecting the eyelids. It is otherwise like malignant pustule, and is mostly soon fatal.

Internal anthrax varies in different cases. The early symptoms are generally restlessness, a sense of depression and exhaustion, and vague sensations in the limbs; then acute fever suddenly sets in with the usual symptoms, and, in addition, great prostration, embarrassed respiration, and rapid collapse. To these may be added the special features of the pulmonary or intestinal forms.

In the *pulmonary* form, difficult and laboured breathing, with a sense of constriction, cyanosis, and great prostration, seem to be the main features, without much cough or physical signs other than a few rhonchi and râles. The expectoration, if there is any, may be bloody. Delirium and coma may precede death, or the mind may be clear to the last.

In the *gastro-intestinal* form there are vomiting, abdominal pain, and diarrhœa, often with blood in the fæces; sometimes dysphagia, and bleeding from the pharynx and mouth. Fever is slight, but dyspnœa and lividity, restlessness, and convulsions of epileptic or tetanic character precede the invariably fatal end.

Anatomical Changes.—In all fatal cases there may be found the changes indicative of acute septic disease: ecchymoses in the submucous and subserous tissues, in the substance of the heart, or in the muscles: hæmorrhage or œdema of the lungs, congestion and softening of the liver and kidneys. The spleen is not always enlarged. When the special *pulmonary* symptoms have been present, there are congestion of the mucous membrane of the trachea and bronchi, hæmorrhages into the lungs or under the pleura, swelling of the cervical and bronchial glands with hæmorrhage into or around them, fluid in the pleural cavities, and ecchymosis and gelatinous exudation in the neck and mediastinum, surrounding the trachea and mediastinal glands.

In the *intestinal* form the peritoneum contains serum, which is often blood-stained; there is semi-gelatinous infiltration of the mesentery and retroperitoneal connective-tissue; congestion, and swelling of the mucous membrane and submucous tissues of the stomach and intestines, in patches of a quarter of an inch to one or two inches in diameter, which are pink and fleshy on section, but on the surface discoloured, or excoriated, or covered with an adherent layer of blood. There are also submucous and subserous hæmorrhages; and the spleen and the mesenteric and lumbar glands are enlarged.

Diagnosis.—Much depends at first on the knowledge of the possibility of infection, especially in the internal forms. With a well-developed malignant pustule, the central eschar and the surrounding ring of vesicles on a red infiltrated base are characteristic. Bacilli may be detected in the fluid from the pustule, or in the blood, expectoration, or urine. But it must be remembered that they are not generally to be found in the blood for some days, though exceedingly numerous in the local sore by the second or third day. The diagnosis may be confirmed by inoculation of a rabbit, guinea-pig, or mouse with the secretions or with blood. The animal dies within two or three days with dyspnœa, dilated pupils, and, perhaps, convulsions; and the blood contains the characteristic bacilli.

Prognosis.—This is very unfavourable in cases left without treatment.

Treatment.—In malignant pustule the most certain cure is to excise the infiltrated part completely, and apply caustic, such as zinc chloride, to the exposed surface. The patient often improves at once, and is soon well. Mr. Davies-Colley, who has treated several cases at Guy's Hospital, finding the bacilli, in the earlier stages, more or less confined to the centre of the pustule, thinks it may not be always necessary to use the caustic after excision. Another treatment, which has been successful in a great many cases, has been the injection of carbolic acid into the tumour. A syringeful (20 or 30 minims) of a 2 per cent. solution of carbolic acid in water is injected into each of four points surrounding the central eschar; and the injections are repeated two or three times a day. In this way sixty or seventy injections have been given in five days, with a good result. Energetic local treatment may still be successful, even when there is evidence of general infection having begun. Internal anthrax should be treated with quinine and carbolic acid, stimulants, and suitable nourishing food.

Mr. Davies-Colley, following the practice of Dr. Muskett of South Africa, has used ipecacuanha externally and internally, with success. He applies ipecacuanha powder to the wound after excision, and gives the drug, internally, in doses of 5 or 10 grains every four hours.

RAG-SORTERS' DISEASE,

Closely resembling internal anthrax in its symptoms and pathological appearances, is a disease which occurs amongst the workers in paper manufactories. A bacillus has been found in the blood, lymph-glands, lungs, and abdominal viscera, which differs in important particulars from the bacillus anthracis, and has been named the *B. proteus hominis capsulatus.*

FOOT-AND-MOUTH DISEASE.

(*Aphtha epizootica.*)

This disease of cattle and sheep is occasionally transmitted to man. The typical feature of the disease in cattle is the formation of vesicles and bullæ on the mucous membrane of the mouth, lips, and tongue. The affected parts become swollen, and the saliva dribbles away. The vesicles break, leaving a grey layer covering the base. Vesicles also appear on the feet round the border of the hoofs, and they become pustular and produce crusts. In cows, vesicles form also on the udders and teats. There is a moderate degree of pyrexia. The disease lasts about a fortnight, and generally ends in recovery; except in calves, of which 50 to 75 per cent die. It is thought that this is due to the milk drawn from the diseased cow irritating the bowel as well as conveying the virus.

The disease appears to be conveyed to man by direct inoculation, and by drinking milk from an infected cow.

The *incubation* is from three to five days. Slight pyrexia and loss of appetite first occur, then vesicles are observed in the mouth, on the lips, tongue, fauces, and hard palate. They reach the size of peas, become opaque, break, and form shallow ulcers, with a dark-red base. The lips become swollen, and saliva and mucus are more abundant than normal. Mastication, swallowing, and [illegible] There may be some diarrhœa and [illegible] fingers, especially about the [illegible] together; and similar [illegible] occurring on the toes, and on the [illegible] is from ten days to a fortnight, [illegible]

[illegible] may be used to the mouth, and [illegible] with solid silver nitrate. Zinc [illegible] should be applied to the eruption on [illegible]

ACTINOMYCOSIS.

[illegible] into the body of a veget- [illegible] Ray fungus. In 1877, Israel of [illegible] cases in man, and in 1878, Ponfick [illegible] human cases with a similar affection [illegible]

[illegible] fungus that is visible to the naked eye as [illegible] grey, glistening, spherical, granular [illegible] -tenth of an inch in diameter, but some- [illegible] -width of an inch, and consisting, under the [illegible] mass of closely woven mycelial threads [illegible] radially, in every direction, multi- [illegible] some of which divide dichotomously, finally [illegible] club-shaped extremities. Although it occurs in [illegible] there is no evidence that it has ever been con- [illegible] man. Animals and men undoubtedly obtain [illegible] source, such as vegetable food; and Bostrom [illegible] the centre of some of the lesions in man [illegible] other cereal, showing almost conclusively [illegible] grain has been the means of the introduc- [illegible] the body. The next stage is the adhesion [illegible] point of the surface of the alimentary or [illegible] possible penetration to deeper parts, and [illegible] lesions in different parts of the body. These [illegible] changes, of more or less intensity, [illegible] granules, so as to form slowly growing [illegible] suppurate, break down, and discharge. [illegible] in the centre the radiating struc- [illegible] immediately around it a thick layer of [illegible] which some of the club-shaped ends of [illegible] and a layer of fibroid connective tissue, [illegible] tumour. From the continued growth [illegible] fungus at one spot—as, for instance, in the liver—large tumours may be formed, three or more inches in diameter, consisting of a kind of cavernous tissue, the trabeculæ of which are fibroid tissue, while the spaces either correspond to the fungus and leucocytes, as above described, or contain pus, in which the yellow granular masses of fungus lie loose. A remarkable feature of the disease is the way in which lesions extend by contiguity from tissue to tissue over long periods of time: but occasionally the fungus seems to be conveyed by the vessels to remote parts, and a more wide-spread deposit occurs.

Symptoms and Course.—These depend on the seat of the primary invasion.

In many cases this is the *mouth*, when a tumour is generally first noticed under the skin over the lower jaw, or on the edge of the jaw. It is hard, does not affect the skin, is chronic in its course, varies in size from time to time, and tends to migrate gradually from the edge of the jaw down to the neck, leaving, for a time, a narrow band of firm tissue in its track. The tumour may shrink up in part, the inflammatory tissue cicatrising, but newer portions continue to form, and ultimately the skin becomes involved, obscure fluctuation is felt, and it opens, discharging a thin, sero-purulent, odourless fluid containing the characteristic granules. A sinus is formed, which rarely closes, but continues patent with slight discharge. There is evidence to show that in these cases the fungus has entered by a carious tooth, and this is held to explain the occurrence of the tumours in connection more frequently with the lower than the upper jaw, and with the back part rather than the front part of the lower jaw. In one case the tumour formed in the substance of the lower jaw, and expanded the bone. Invasion by the upper jaw results in tumours of the cheek or temple; and an extension to the base of the skull or the mediastinum by means of the pharynx is a possibility which makes the implication of the upper jaw more serious than that of the lower. Penetration of the *œsophagus* has led to mediastinal abscesses, and erosion of the vertebræ.

In a case of *intestinal* actinomycosis, the mucous membrane presented on its surface patches of whitish material, covered with yellow and brown granules. The patches were about two-fifths of an inch in diameter and one-fifth of an inch thick, and adhered firmly to the membrane. The disease may also cause swellings in the substance of the intestinal wall, from which it may perforate into the peritoneal cavity, or, by means of adhesions, invade adjacent viscera or the abdominal wall at almost any point. The *liver* is often secondarily infected in intestinal cases, and then contains large masses or even prominent tumours, having the structure above described. Clinically, such tumours may present the characteristics of hepatic abscesses, with local pain, tenderness, remitting fever, and rigors.

When actinomycosis affects the *lungs* the symptoms may be bronchitic or pneumonic. In a case of the former kind there was a close resemblance to putrid bronchitis, the sputum separating into two layers (not into three, as in fœtid bronchitis), the upper clear, and the lower turbid; the latter contained the ray-fungus. When the lung substance is affected, pneumonia occurs in patches, the patients cough and lose flesh, and the expectoration is either thick and muco-purulent, containing the typical granules, or it may be viscid, translucent, and rusty, like pneumonic sputa. There is often a certain resemblance to phthisis, but the posterior and lateral portions of the lungs are involved, not the apices; and the sputum is, of course, free from tubercle bacilli. If the inflammatory lesions reach the surface they set up pleurisy or pericarditis. Effusion takes place, or the lung becomes adherent to the chest wall, which then becomes involved and ultimately soft diffused inflammatory swellings appear on the chest, which may fluctuate, break, and discharge purulent fluid containing the fungus. From the lung, also, the inflammatory track of the fungus may stretch through the diaphragm into the abdomen, or behind the diaphragm to the psoas and iliacus muscles. These processes are commonly very slow, and are accompanied with varying amounts of fever in different cases.

The *skin* was involved in a case recorded by Pringle; there were large, soft, fleshy, sarcoma-like outgrowths of mottled purplish-red and yellow colour, covered by very thin skin, and presenting small ulcerative openings, from which a sticky fluid oozed, and in which lay a purulent fluid containing actinomyces granules.

Diagnosis.—This can only be made with certainty by the detection of the characteristic granules in the secretions, whether pus, sputum, or urine. Some care is required in looking for them. Pus may be shaken up with a little salt and water in a test-tube, when the granules will come into view. One of these may be then placed on a slide and covered with thin glass, when a low power will show the characteristic radiating structure. The inflammatory lesions are said to present a specially "wooden" resistance.

Treatment.—Complete extirpation of the growth, or scraping out the resulting abscess or sinus, so as to completely remove all fungus granules, has cured some cases, in which the lesion was confined to the neighbourhood of the jaw. When the viscera are involved there is less hope of recovery; but the chances are somewhat better in the case of the abdomen than of the chest, since the latter is more accessible to surgical help. In any case *iodide of potassium* should be given to the extent of two drachms daily, as very remarkable results have been obtained under its use both in bovine and human cases.

DISEASES OF THE NERVOUS SYSTEM.

UNDER this heading we have to deal with disorders of the brain, spinal cord, and nerves—disorders which manifest themselves through the functions of motion, sensation, the special senses, and the intellect and emotions.

The nervous system is liable to similar lesions with the rest of the body. It has been already shown to suffer from the toxins of infectious diseases, either in common with the rest of the body, as in typhus and enteric fevers; or more apart, as in tetanus, hydrophobia, beri-beri, and leprosy. Still more localised inflammations and degenerations may take place in the nervous system, tumours may grow in various situations, or the different parts may be crushed or injured. And it is a consequence of the specialisation of almost every nerve and nerve-centre for a particular function, that when damage is done by injury, inflammation, or tumour, the symptoms it produces depend very much, or entirely, upon the precise *locality* in which it occurs. The effect of a patch of pneumonia in the lung is very much the same whatever part it involves; but the effect of a limited lesion in the nervous system may be for a long time harmless in one spot, and quite rapidly fatal in another. The lung subserves mainly one function; the brain and spinal cord a great number. It is the *localisation of functions* in different parts of the nervous system that enables us to determine the position of the *disease* when it occurs; and as, for this purpose, in the case of the lungs we examine every part of the surface of the chest by percussion and auscultation, so in the case of the nervous system we investigate every function—the power of motion in every part of the body, the accuracy of sensation, the perfection of the special senses, and the integrity of the intellect and emotions. Having ascertained the seat of the lesion, we may, from our knowledge of the diseases that affect certain localities, make a complete diagnosis; on the other hand, we may sometimes, from the nature of the attack, or from points in the patient's history, be enabled to recognise the nature of the lesion, when its exact localisation is still uncertain.

Before proceeding to the systematic description of the diseases of the nervous system, I must say something of its general anatomy and of the clinical methods of investigating the symptoms which its diseases produce.

GENERAL ANATOMY.

The organs of the nervous system are made up of what have been hitherto called nerve-cells and nerve-fibres, united together by connective tissue, a special form of which in the brain and spinal cord is known as *neuroglia*. Recent researches have shown that we must regard every nerve-fibre as being associated with a nerve-cell, so as to form a separate structure, the *neuron*. And practically the whole nervous system is made up of neurons, of which at least some are motor and others sensory. A neuron consists of: (1) a *cell-body*, with a large nucleus and nucleolus; and the nutrition of every part of the neuron depends upon its connection with the cell-body and the integrity of the nucleus. (2) An *axis-cylinder process*, hitherto known as an axis-cylinder. This is smooth, uniform in diameter, runs for long distances as nerve-fibre, joining with others to form nerve-trunks; it gives off branches from time to time, called *collaterals*, and terminates in tufts of fibrils, called *arborisations*. The terminations and collaterals end absolutely free about the cell-bodies, or other tufts or endings, but never unite or anastomose with them. (3) One or more other processes, called *dendrons*, which break up into branches not far from the cell-body, do not have axis-cylinders, are often rough on the surface, and also break up into free extremities.

The motor functions of the nervous system are subserved by two groups of *motor neurons*. The *upper* or *central motor* neurons have their cell-bodies situated in the cortical motor area of the brain, forming the well-known pyramidal cells; they have a few dendrons in the immediate neighbourhood, and they send long axis-cylinder processes downwards which form the pyramidal tract, cross the middle line (decussation), occupy the crossed pyramidal tracts of the spinal cord and terminate by arborisations which interlace with the dendrons of the neurons of the other group. This comprises the *lower* or *peripheral motor* neurons; their cell-bodies are situate in the anterior cornua of the spinal cord and the corresponding nuclei of the medulla oblongata and pons, their axis-cylinder processes pass out into the motor nerves, and their final tufts or arborisations come into relation with the end-plates of the muscles.

The groups of sensory neurons are three in number. Of the *lowest* or *peripheral* neurons the cell-body is situate in the ganglia on the sensory roots of the nerves. They have an axis-cylinder process, which after a short distance divides into two branches. One of these goes downwards in the nerve trunk to be connected with the skin and muscle-spindles, and is regarded by some, not as an axis-cylinder, but as a protoplasmic process or dendron. The other branch passes upwards into the cord, where again it divides, one branch passing downwards, the other upwards. Both send off collaterals, some of which surround the cell-bodies of the lower motor neurons in the anterior cornua; others, the cell-bodies in Clarke's column, of which the axis-cylinder processes pass up in the cerebellar tract of the same side; others, the cell-bodies of other neurons, of which the axis-cylinder processes cross the middle line and pass up the cord in the opposite antero-lateral ascending tract. The ascending branches of the main axis-cylinder pass up the spinal cord and come into relation with the *second* sensory neuron; of which the cell-bodies are situate in the medulla oblongata as the nuclei of the posterior columns, *nucleus gracilis* and *nucleus cuneatus;* while their axis-cylinder processes cross the middle line in the decussation of the fillet, join the processes from the antero-lateral ascending tract, and pass up towards the cortex. In the thalamus opticus they terminate around the cell-bodies of a *third* or *upper* sensory group of neurons, of which the axis-cylinder processes pass up to the cerebral cortex, when they come into relation more or less directly with the cell-bodies of the upper motor neurons. Some of the sensory axis-cylinder processes pass upwards in the posterior part of the internal capsule.

There are, of course, many other kinds of neuron, subserving the special senses and other functions.

The general results of lesions of the neurons may be considered. *Destructive lesions.*—The lesion of any neuron causes a degeneration of that portion which is cut off from the nucleus of the cell-body, and hence, lesion of the cell-body itself destroys the whole neuron. Lesion of the upper motor neuron (cortical motor area or pyramidal tract) causes secondary degeneration or sclerosis of the axis-cylinder processes below the lesion (*see* Secondary Degeneration), but leaves the lower neuron intact. Lesion of the lower motor neurons causes degeneration of axis-cylinder processes below the lesion, that is, of nerve-trunks; and as the muscles with which the neurons are connected depend upon them for their nutrition, they become degenerated or atrophied as well.

Functionally the result of destructive lesions on either motor neuron is paralysis to voluntary impulses of the muscle in which the lower of the two terminates. If the upper neurons in the brain are alone affected, the paralysis is on the opposite side of the body; it involves as a rule a large number of muscles, such as the arm or leg, or the whole side of the body, and not single muscles: and spinal reflex action remains undiminished. If the lower neuron is alone affected, the paralysis is on the same side of the body as the lesion. Single muscles may be picked out or a whole limb involved according to the number of neurons diseased, spinal reflex action is abolished, and the nutrition and electrical reactions of the nerves and muscles concerned are lost or impaired.

So-called *irritative* lesions of the upper motor neuron in the cortical area cause spasmodic contractions of the muscles with which they are in connection on the opposite side of the body. Those of the lower motor neurons are less constant in their results. Some spasms in spinal meningitis and the fibrillary twitchings of progressive muscular atrophy may be due to them.

Lesions of the sensory neurons cause various modifications of sensation, and with severe lesions this function is abolished completely. Under other circumstances pain may be the result. Related to injury of the lower sensory neurons are certain lesions of nutrition of the skin and joints, such as are seen in locomotor ataxy and peripheral neuritis.

CLINICAL EXAMINATION OF THE NERVOUS SYSTEM.

MOTOR SYMPTOMS.

These consist of *paralysis*, *spasm* or *convulsion*, and *inco-ordination.*

Paralysis, or loss of power in the muscles, is mostly due to lesions of the nervous system; but it occurs in some diseases of the muscular substance itself, such as pseudo-hypertrophic paralysis. Loss of power may also arise from defective nutrition, such as follows prolonged illness; but it is not then included under the term paralysis. In all forms, the weakness may be of any degree up to complete abolition of movement. The lesser degrees are sometimes called *paresis* or *partial paralysis.* Paralysis in the upper limb may be tested by getting the patient to grasp one's hand with his, by forcibly extending (or flexing) his arm, which he tries to keep flexed (or extended); in the lower limb by his power to stand upright, or to raise the heels from the ground, or to carry a weight on the back, or, while sitting on a chair, to flex or extend the leg against the observer's pressure; or, if in bed, to raise the foot and leg from the bed, or to draw the knee up to the abdomen, or to raise the leg while the observer presses upon the knee. Comparison should be made with the limb of the opposite side, or with those of a healthy individual of the same sex, age, and muscular development. The grasp of the hand can be usefully tested by the *dynamometer*, which, in its usual form, consists of an oval steel ring 5 by 2½ inches, which, when compressed laterally, registers the extent of movement on a dial, scaled to pounds. It can also be used by pulling on the two ends.

Names have been given to special forms of paralysis, such as *hemiplegia*, paralysis of one side of the body; *paraplegia*, paralysis of the legs, or the legs and trunk; *monoplegia*, paralysis of one limb, &c.

Spasms, or *Convulsions*, are involuntary muscular contractions, which may be (1) interrupted, or *clonic*; (2) continuous, or *tonic*.

In the former there are quickly alternating contraction and relaxation of the muscles, producing jerking or *tremor* or more violent to-and-fro movements of the limb, or of whatever part is affected.

Intermittent

In the latter the contraction is continuous while it lasts—that is, there are no quick changes in the condition of the muscle. If it is persistent, it is called *contracture;* and in very long continued cases, structural changes take place in the muscle which hinder recovery.

Continuous

In some cases of localised paralysis, permanent contraction results in the antagonist muscles.

Inco-ordination.—Muscular movements, especially the more complicated, require an exact adaptation in the contractions of the different muscles concerned. These are not only the muscles primarily needed to effect the movement, but also their antagonists; and, if this adaptation is imperfect, the movement becomes irregular, or disorderly, and *inco-ordination*, or *ataxy*, results. It may be tested by asking the patient to walk along a straight line, when the gait is found to be staggering, or reeling, or waddling; to turn quickly when walking; to lift the heels from the ground when standing; or to stand with the eyes shut and the feet close together (Romberg's symptom). In the arms it may be obvious on the patient putting out his hand to seize objects; or slighter degrees may be brought out by the patient shutting his eyes, and then trying to touch his nose with the tip of his forefinger, or to hit a spot on a sheet of paper with a pencil.

SENSORY SYMPTOMS.

The sensations of touch, temperature, and pain are not always affected together, and each should be separately examined.

Anæsthesia, or loss of tactile sensibility, can be tested by lightly touching the surface of the skin with a feather, or a strip of paper, while the patient's eyes are closed. He should be able to say *when* and *where* he is touched. Fallacies are common, and the result must be checked by frequent trials. Some idea of the extent of anæsthesia may be gathered by noting the distance at which two points touching the skin can be recognised as two, or are thought to be only one. In the most sensitive parts they are recognised as two when less than 2 mm. apart; in the least sensitive the distance must be 2 or 3 inches. The *æsthesiometer* is an instrument devised for the accurate measurement of these points; it consists of a graduated bar, on which two points can slide easily, so as to be fixed at a required distance from each other. The following are some of the measurements given by Weber, representing the distances at which the points can be distinguished as two, under normal conditions:—Tip of the tongue, 1·5 mm.; finger-tips, 2 to 3 mm.; the lips, 4 to 5 mm.; the cheeks, and backs of the fingers, 12 mm.; the forehead, 22 mm.; the neck, 34 mm.; the forearm, leg, and dorsum of foot, 40 mm.; the chest, 45 mm.; the back, 60 mm.; the upper arm and thigh, 75 mm. Leube has proposed to move a point along the skin, and observe the least amount of movement that can be recognised as such.

A touch may be distinctly felt, but an unusually long interval may take place between the stimulus and the patient's recognition of it; this is *delayed conduction*.

Hyperæsthesia is the production of pain from impressions commonly painless. **Paræsthesia** and **dysæsthesia** are terms used to designate those modifications of tactile, as well as painful, impressions, which result in "tingling," pricking," "pins and needles," &c., and also various subjective sensations, as formication and the numb feeling. Sometimes a touch is felt as if it were two, three, or more (*polyæsthesia*), or it may be felt in another part of the body, or at the corresponding part of the opposite side of the body (*allochiria*).

Analgesia means insensibility to pain. It may be tested by pinching, or by pricking with a blunt point, such as the point of a quill pen; or by applying the faradic current with wire brush terminals. *Hyperalgesia* may be used for an excessive sensibility to painful impressions.

Sensibility to pressure on the skin can be tested by weights applied to it, and the minimum variation of the weight that can be recognised must be noted. In health it is about one-twentieth of the total pressure.

Sensibility to temperature may be tested by the application of hot and cold spoons, a spoon being heated by immersion in hot water; or by the application of test-tubes holding hot and cold water. Differences of temperature may be entirely unrecognised; cold may be taken for heat, or the reverse. Probably sensations of heat and cold are conveyed by different nerves.

Muscular sensibility is commonly tested by asking the patient to distinguish between different weights placed in his hand. It is important that no clue should be given to the sense of sight or the sense of touch; hence the weights should be of the same size and shape, or, better, they should be placed in a bag, suspended by a string. Other methods are the power of recognising the position into which the limb is put, the sensitiveness of the muscles to deep pressure, and to electrical stimulation. In the first of these cases, cutaneous sensation must be eliminated by firmly grasping the limb at the point where it is held; and by the injection of cocain in the last.

REFLEXES.

Two groups of reflex action, partially familiar to us for many years, have been carefully investigated, and applied to the diagnosis of nervous diseases. One group is that of *superficial* or *cutaneous reflexes;* the other, that of *deep* or *tendon reflexes.*

Cutaneous Reflexes.—When the skin of the inner side of the thigh is slightly scratched or stimulated, the cremaster muscle of the same side contracts and draws up the testicle. This is a purely reflex action, depending upon the integrity of an afferent and a motor nerve, and of the nerve-centres in the segment of the spinal cord with which they are connected: it is called the *cremasteric reflex.* There are other parts of the surface where stimulation readily elicits muscular contraction; thus, on stimulating the sole of the foot, the foot and leg are drawn up—*plantar reflex;* a *gluteal reflex* is obtained by stimulating the surface of the buttock; an *abdominal*, by scratching the abdomen along the outer border of the rectus; an *epigastric*, by irritation over the cartilages of the lower true ribs; a *scapular*, by stroking the skin between the shoulder blades, when the attached muscles contract. Since these movements require the integrity of the reflex arc, they may be abolished by disease of the centre, of the afferent or of the efferent nerve; and it is important to know the segment or segments of the spinal cord to which each *reflex* corresponds. These relations are given in the table which will be found under "Diseases of the Spinal Cord."

Some facts in connection with these reflexes in diseases of the brain and spinal cord seem to justify the hypothesis that the skin reflex centre in the spinal segment is controlled by a centre situated in the brain, and this again by a third centre situated in the cortex. The facts are, first, that in spinal lesions cutaneous reflex is exaggerated in the parts corresponding to the cord below the lesion; and secondly, that in hemiplegia from cerebral disease the skin reflex is diminished on the side of the paralysis.

Deep Reflexes.—When the leg is hanging freely, with the knee bent at a right angle, and the ligamentum patellæ is sharply struck with the tips of the fingers, the ulnar edge of the hand, the edge of a book, or other similar object, the rectus femoris contracts, and the foot is jerked sharply forward. This has been called the *knee-phenomenon*, *patellar-tendon reflex*, *patellar reflex*, or *knee-jerk*. It is generally well obtained, in the sitting position, by crossing one leg over the other and striking the upper knee. Often it can be obtained by striking above the patella. When the reaction is slight, the patient should sit on a table, with the legs hanging over the edge, and the knees should be bare. If it is not elicited then, the patient's attention should be distracted, as by getting him to hold the fingers of one hand in those of the other, and look up to the ceiling while he pulls at his hands (*reinforcement*). When the patient is in bed, the leg may be raised by placing the hand under the knee; or, the patella being pushed down by a finger placed across the top of it, this finger is struck with the fingers of the other hand (*depressed patellar reflex*). When obtained in this way, it is generally regarded as exaggerated. *Ankle-clonus* or *foot-clonus* is a similar phenomenon, which occurs in certain spinal and other diseases, but is not, like the knee-jerk, present in health, except in a modified form. To elicit it, the patient should be in bed or in a chair; the leg is lifted with the left hand, and the foot, held firmly by the toes in the right hand, is forcibly bent towards the knee. Immediately the calf muscles contract, but as the pressure on the foot is maintained, they relax, again contract, and so alternately contract and relax for an almost indefinite period, constituting the so-called *clonus*. When the flexion of the foot fails to start the contractions, they may be brought out, while the foot is flexed, by a tap on the front of the leg (front tap), or on the tendo Achillis. The contractions occur at the rate of about seven in a second. The modification of this phenomenon that occurs in health is the series of rapid alternating movements which can be kept up continuously and without effort when, in the sitting posture, the foot rests upon the ground by the tips of the toes only. Reflexes, similar to the knee-jerk, can be sometimes obtained with the following tendons:—tendo Achillis, adductors of the thigh, triceps, supinator longus, extensors at the wrist, and jaw; and in some cases a *clonus* can be obtained at the knee.

The phenomenon of the knee-jerk was first spoken of as tendon-reflex, under the idea that an impression on the tendon was transmitted to the spinal cord, and reflex contraction of the rectus muscle followed. Against this are the facts, first, that the interval between the stimulus and the movement is much less than that required for a spinal reflex from the skin of the knee; and secondly, that the knee-jerk takes place when the patellar tendon nerves are divided. The explanation commonly adopted is that the stretching of the muscle, by bending the knee, reflexly increases the tone of the muscle, so that a local stimulation readily excites its contraction. As the tone depends upon the cell-bodies of the lower neuron, the integrity of the reflex is necessary for the occurrence of the knee-jerk. In foot-clonus the forcible stretching of the calf muscles not only gives the required tension, but also acts as a local stimulant to set up contraction; and, the tension continuing, each contraction is rapidly succeeded by another. The segments of the cord concerned in the production of knee-jerk are those which correspond to the second, third and fourth lumbar nerves; of ankle-clonus, to the fifth lumbar and first sacral nerves.

Paradoxical contraction of Westphal is a slow contraction in a muscle when it is suddenly and passively shortened. Thus, if the foot be bent up towards the knee, the tibialis anticus will after an interval contract, and maintain the foot bent for a time and then slowly relax. Its significance is not known.

CHANGES IN NUTRITION.

The nutrition of the tissues is profoundly affected in some diseases of the nervous system, but there is no evidence of separate trophic nerves or neurons. The most marked effects are seen in lesions of nerve-trunks and their centres, *i.e.*, those which injure the lower neurons, motor and sensory. Thus, lesions of the anterior cornua (infantile paralysis) or of the nerves (injury, neuritis) are accompanied with marked wasting of muscle, which is not present in lesions of the brain or cord, involving the upper neurons only. Wasting of muscle is first shown by flabbiness; later by actual diminution in size. Its extent can be estimated by measurement, but it must be remembered that subcutaneous fat may completely mask a good deal of wasting, so far as bulk is concerned. Other parts besides the muscles are often involved. The skin in some chronic cases becomes thin, red, and shiny—the "glossy skin" of Paget; erythematous, bullous and vesicular eruptions (*e.g.*, zona), œdema, whitlows and ulceration of the skin may occur; the finger-ends are pinched, from wasting of the subcutaneous tissue; the growth of hair and nails is retarded; and the nails are brittle. The bones may also suffer in their nutrition, becoming brittle or breaking easily; and if paralysis occurs in early life, growth of a whole limb may be retarded, so that it is eventually one and a half to two inches shorter than its fellow. In acute cases, the temperature of the skin is raised, the vessels dilate, vesicles or bullæ form, and bed-sores occur on the slightest irritation or pressure.

ELECTRICAL CONDITIONS.

Two forms of electricity are commonly used in medicine: the *faradic*, *induced*, or *interrupted* current, derived from an induction coil; and the *galvanic*, *voltaic*, or *continuous* current, derived from several cells, numbering from two to forty or fifty, joined in series—that is, with the positive plate of each in connection with the negative plate of its neighbour. In the former the current is alternately *closed* and *opened* (*made* and *broken*) with great rapidity by the mechanism employed; in the latter the closing and opening of the circuit are commonly effected, much more slowly, by the hand.

The Faradic Current.—This is best applied by placing the two terminals at no great distance from one another on the surface of the muscle to be stimulated or on the nerve which supplies the muscle. It is indifferent which pole is uppermost (nearer the nerve centres), as the current passes alternately each way. The stronger the current, the greater is the contraction. The faradic current causes contraction by stimulating the nerve-trunks and the nerve-endings in the muscles, but not the muscular substance itself. Consequently, when the nerve is injured or degenerated, or cut off from its nerve-centre (lesion of lower motor neuron), the reaction to faradism is completely lost, both on application to the nerve and to the muscle.

The Galvanic Current.—This is applied by placing one pole, or terminal, on the spine, the other on the nerve or muscle to be tested. The results are different, according to the position of the poles—that is, whether the kathode, or negative pole, is on the muscle, and the anode, or positive pole, nearer the centre, or *vice versâ*. In the former, the current is said to be *descending*, *direct* or *kathodal*; in the latter, *ascending*, *inverse*, or *anodal*. A galvanic current of moderate strength causes no contraction while the circuit is complete—that is, while the two terminals are continuously applied; but contraction takes place (1) when the circuit is broken, for instance, by lifting the terminal from the muscle, and (2) when the circuit is completed again. These contractions are partly due to stimulation of the nerve-endings, but partly also to stimulation of the muscular substance itself; and in certain stages of nerve injury and degeneration, when the faradic current fails to elicit any reactions, and the galvanic current applied to the nerve gives no result, the latter current applied to the muscle calls forth contractions, the characteristic of which is that they are slower and longer than under normal circumstances.

Polar Reactions.—Some important results have now to be pointed out, which are explained by the facts of electrotonus.

It has been already stated that the galvanic current may be direct or inverse, and that the contraction takes place when the current is either closed or opened. This gives four different conditions of contraction: (α) Closing the direct (kathodal) current, (β) opening the direct, (γ) closing the inverse (anodal), and (δ) opening the inverse. These contractions are generally indicated by symbols, as follows:—

- (α) KCC, Kathodal Closure Contraction, meaning the contraction which takes place when the circuit is *closed* with the kathode or negative pole on the muscle (hence a descending current).
- (β) KOC, Kathodal Opening Contraction, or contraction when the current is *opened* with the kathode on the muscle.
- (γ) ACC, Anodal Closing Contraction, or contraction when the current is *closed* with the anode or positive pole on the muscle (hence an ascending current).
- (δ) AOC, Anodal Opening Contraction, or contraction when the current is *opened* with the anode of the muscle.

If, in health, the attempt be made to ascertain what is the smallest number of galvanic cells—that is, the strength of battery proportionately—which will cause the four kinds of contractions, it will be found that KCC requires the smallest number, often six or eight of an ordinary battery; ACC requires more; AOC as many or sometimes more than ACC; and KOC is only brought out by a very strong current, or by none at all that can be borne by the patient. Moreover, currents that are required to bring out strong contractions with AC (Anodal Closure) or AO (Anodal Opening) produce tetanic contractions with KC (Kathodal Closure).

The ascending order represented by the following formula should be remembered—

KCC
ACC
AOC
KOC

Or in more detail as follows:—

Strength of Current.	Kind of Contraction.
Very weak . .	KCC only.
Weak . . .	KCC stronger, ACC, and AOC.
Moderate . .	KC tetanic, AC and AO stronger contractions.
Strong . . .	KC strong tetanus, AC and AO stronger contractions, KOC.
Very strong . .	KC very strong tetanus, AC tetanus, AO strongest contractions, KOC stronger.

Thus, for the same amount of current strength—

$$\text{KCC} > \text{ACC},\ \text{ACC} = \text{or} > \text{AOC},\ \text{AOC} > \text{KOC}.$$

In various forms of degeneration of nerve and muscle, these relations are often altered—KCC requires as many or more cells than ACC, and AOC as many or more than KOC. Thus, instead of the order of excitability being—

KCC
ACC
AOC
KOC

ACC may be equal to KCC, thus:—

$\left.\begin{array}{l}\text{KCC}\\\text{ACC}\end{array}\right\}$
AOC
KOC,

or ACC may exceed KCC, and KOC may exceed AOC, thus:—

ACC
KCC
KOC
AOC.

These alterations are described as *qualitative* changes, to distinguish them from simple increase or decrease of susceptibility to the current, which may be called quantitative changes. The former are also called *polar* changes.

Reaction of Degeneration.—The most important indication of disease affecting the nerve-nuclei, nerve-trunks, or muscles, is derived from the use of both currents, and is known as the reaction of degeneration. The symbol RD may be used. *Complete* RD consists of loss of excitability in the nerve to both faradic and galvanic currents; loss of excitability in the muscle to the faradic current; increased excitability of the muscle to the galvanic current, with *qualitative* or polar changes, and sluggish contractions. In *incomplete* forms of RD the reaction of the muscle to galvanism is as just described, but the excitability of the muscle to faradism, and of the nerves to either current, is not lost, but only lowered. A *mixed* form is also described, in which the muscle-contractions to galvanism are neither very sluggish nor very prompt, and ACC may not exceed, or may even be less than, KCC. It occurs when degenerated fibres of muscles or of nerve lie side by side with others which are healthy.

Measurement of Current.—In recording the amount of galvanism used for testing the nerves and muscles, it is very desirable that a galvanometer should be used, and that the amount of electricity employed in each operation should be recorded in *milliampères.* The number of cells forms no certain guide to the quantity, as their electromotive force, and the resistance of the tissues through which the current passes, vary much from time to time.

Motor Points.—There are a number of points on the surface of the body, where the nerves and muscles are especially accessible to electrical stimulation; and on these points the rheophore should be placed, if the maximum contraction of any muscle is required. Thus, for the long head of the triceps there is a point close up to the axilla; for the internal head there is one midway down the arm on the inner side; for the brachialis anticus the point is a little below the middle of the arm, at the inner border of the biceps muscle. These are called Ziemssen's motor points.*

SPECIAL SENSES.

The special senses may have to be investigated in any case of nervous disease, as they may give important information as to the condition of the centres.

Hearing may be tested by the tuning-fork, or the watch: and the auditory meatus should be examined with the speculum.

Smell.—One nasal orifice may be closed, while with the other the patient is directed to smell such substances as musk, valerian, essential oils, eau de Cologne, or camphor. It must be remembered that ammonia and other pungent substances irritate the nerves of touch rather than those of smell.

Taste may be examined by the application of salt, sugar, or quinine, to each side of the tongue alternately.

Vision is affected in several ways in nervous diseases. The size of the pupils should be noted, and the two pupils compared. It should be noted whether they contract to light, or during accommodation. The patient's power of vision for near objects and distance should be inquired into, and the presence of myopia or hypermetropia determined. Inability to see objects closer, while they can be seen at a distance, is due, in old persons, to slow changes in the lens (presbyopia), in young persons to paralysis of accommodation, which may occur rapidly, as after diphtheria. *Diplopia* or *double vision* is mostly the result of paralysis of the external ocular muscles. The *field of vision* may have to be tested, as some patients have good central vision, and are quite unaware of important defects in the rest of the visual field. This may be done roughly by asking the patient to look steadily at an object about a foot in front of him, and then bringing objects gradually within his field, from above, below, and either side, and noting when he can first see them. Each eye should be tested separately with the other closed. More accurate results can be obtained with an instrument called the *perimeter*. Valuable information is gained in many nervous diseases by an examination of the optic disc with the ophthalmoscope. The change of most importance to the physician is inflammation of the optic disc

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* Illustrations of them are given in several works on Therapeutics and Medicine: Erb, H. C. Wood, Hale White, Bristowe, Strümpell, Finlayson, Vierordt.

(*optic neuritis* or *papillitis*). In the early stages the disc is more vascular, red instead of pink in colour, the edge is blurred, indistinct, and changes its thin sharp outline for a broad violet or purplish zone; the retinal veins become tortuous. In later stages, the disc looks larger than normal, the surface is red or purplish-red in colour, with an appearance of radial striation; the veins, as they lie on the retina, are markedly tortuous, full, and dark, whereas on the disc itself they are partly or entirely concealed by effusion; the arteries are thin. Subsequently the disc becomes more prominent; it may be mottled white and red by effusion of lymph and blood respectively, which quite conceal the retinal vessels, while the disc vessels are increased in number and size; or the effusion may be so great as to form a prominent button of grey colour, from which proceed the very thin retinal arteries, and full tortuous veins. After three or four months the lymph is gradually absorbed, and atrophy takes place, producing a small disc of a bluish white colour, with small retinal vessels (*optic atrophy*). It is important to notice that in early stages of optic neuritis ordinary vision is commonly retained, so that the patient may be quite unaware of anything wrong with his eyes, and the lesion is only detected on examination with the ophthalmoscope; in such cases the visual field may be found smaller than normal. With the highest degrees of neuritis and with atrophy, vision is lost or reduced to the mere perception of light.

DISEASES OF THE NERVES.

NEURITIS.

Inflammation of the nerves, or *neuritis*, arises from direct injury, such as blows, punctured or lacerated wounds, over-stretching, the pressure of bones in fractures and dislocations, and compression by the action of muscles through which they pass. Inflammation in the neighbourhood of nerves may extend so as to involve them: and this may happen in suppurating joints, in osteo-arthritis, in pleurisy affecting the intercostal nerves, or in cerebral and spinal meningitis. Cold is a frequent cause of neuritis, which is then often called rheumatic; and acute general diseases, like enteric fever, small-pox, and diphtheria, are sometimes followed by it. The last, as well as many other conditions affecting the system generally, produce, as a rule, a multiple neuritis, and are enumerated under that head (*see* p. 179).

Neuritis may be ***interstitial***, affecting mainly the connective tissue; or *parenchymatous*, affecting first the nerve-fibres themselves. In acute neuritis the nerve is red and swollen, there may be small hæmorrhages, and the microscope shows leucocytes infiltrating the sheath and the septa between the bundles of nerve-fibres. If these changes are limited to the sheath or perineurium (*perineuritis*) the nerve-fibres may escape any serious lesion; but if it is interstitial they are more likely to be affected, the myelin becoming atrophied, and the axis-cylinders suffering less. Ultimately a new fibrous tissue is developed in the interstitial tissue and sheath. In the parenchymatous form the disease appears to begin in the nerve-fibres. The myelin of the white substance of Schwann is broken up into fragments and globules, the nuclei of the internodal cells enlarge and divide, and the protoplasm increases in quantity or becomes granular. The axis-cylinders, at first breaking into segments with the myelin, disappear, while the myelin, becoming less and less, may leave the nerve-tubes nearly empty, containing only here and there nuclei, some finely granular matter, or brownish pigment granules. The nerve-fibres are generally affected unequally.

Secondary Degeneration.—Injuries, which cut off nerves from their centres, cause important alterations below the lesion. These have been closely studied in animals, and similar changes follow neuritis, as well as direct injury, in man. They consist of a degeneration of the nerve-fibres, and atrophy or degeneration of the muscles supplied by them.

The nerve-degeneration known as "secondary degeneration," or "Wallerian degeneration," from the physiologist who first described it, is very similar in its nature to the parenchymatous inflammation just mentioned; and it may be accompanied by varying degrees of interstitial change. In rabbits the first complete interruption of the myelin and axis-cylinder takes place about the second day after the lesion, but in man more slowly, probably from the fourth to the eighth day. The change takes place simultaneously along the whole length of nerve below the lesion.

More or less perfect regeneration of the nerve-fibres may take place most readily in slight lesions; and this begins at the central end by the growth of new axis-cylinders, which afterwards become covered with myelin.

The muscles in connection with such injured nerves become flabby, and lose bulk (*muscular atrophy*, *amyotrophy*). The muscular fibres diminish in size, the transverse striation becomes less distinct, and the substance granular. Later, the transverse striation is lost or replaced by longitudinal striation, and there is an increase of the connective tissue between the fibres.

Electrical Reactions.—The reaction of degeneration (p. 174) follows the lesions above described. Faradic irritability in both nerve and muscle diminishes rapidly, and may disappear by the end of the second week. Galvanic irritability in the nerve disappears in about the same time. But the irritability of the muscle to galvanic currents, though at first less than normal, in a few days becomes excessive, and remains in an exalted condition for some weeks, then gradually falls to the normal, or even, for a short time, below it again. Qualitative or polar changes also generally occur, and complete the reaction of degeneration.

In recovering cases, the faradic irritability of nerve and muscle, and the galvanic irritability again appear about the eighth or ninth week (Erb), and gradually attain the normal. The return of power in the paralysed part generally precedes any decided improvement in the electrical reactions. In severe lesions with extensive atrophy of muscle RD persists for some time, but after some weeks the irritability of the muscle to galvanism also diminishes, and finally becomes extinct.

Some variations in the phenomena occur, especially in the slighter forms of lesion. Thus, the usually early fall of irritability may be delayed some days; or the initial fall of the irritability of muscle to galvanic currents may be absent, and the increase may be delayed some days; or, in very slight cases, the irritability of the nerve to both faradic and galvanic currents may be for a few days increased.

Symptoms.—Neuritis and resulting degeneration involve the paths of conduction for motor and sensory, trophic and vasomotor impulses. There is paralysis, followed by flabbiness and atrophy of the muscles, often with tenderness to pressure, or on contraction. Diminution or loss of sensation occurs where sensory fibres are involved, though this is often, in mixed nerves, very small in proportion to the paralysis. Increased sensitiveness, or pain, or hyperæsthesia may be present in the distribution of the nerve, and pain or tenderness of the nerve-trunks. The cutaneous and deep reflexes cannot be elicited.

The trophic and vasomotor changes above mentioned (p. 171) mostly result from nerve degeneration.

In acute forms of neuritis there is more or less pain in the nerve itself, and in the part to which it is distributed. The pain is worse at night, and increased by movements or positions that cause stretching or pressure on the nerve. If the nerve-trunk can be felt, it may be found swollen and tender, and the skin may be red over it, or even œdematous; but this is rare. Tingling and hyperæsthesia may be also present. Later on, sensation may be diminished, and the muscles may present twitchings or cramps at the same time losing power and becoming tender. Ultimately they atrophy and give the reaction of degeneration. Slight constitutional disturbance may accompany the onset, but it soon passes off.

In chronic neuritis pain is an early symptom, and constitutional disturbance is absent. Changes in sensibility, atrophy of the muscles, degenerative reaction, glossy skin, and other nutritional defects follow.

The duration of neuritis is very variable: slight cases may recover quickly, others last weeks or months. Sometimes neuritis spreads upwards, and, reaching a plexus, invades the several nerves proceeding therefrom. This is called *migrating neuritis*. It may extend to the spinal cord, setting up myelitis or meningitis, and to some such process Gowers is inclined to refer the forms of paralysis which follow visceral disease (*e.g.*, cystitis), and are known as "reflex paralyses."

Diagnosis.—Neuritis must be recognised by its symptoms—sensory, motor, and trophic—being limited to the distribution of a nerve, which is, at the same time, painful and tender. In its early stage it may simulate rheumatism or ostitis, and, from the pain alone, it may be mistaken for neuralgia, in which, however, anæsthesia and motor paralysis do not occur.

Treatment.—The first indications are to remove the cause, if possible, and keep the affected part at rest in such a way as to avoid all irritation of the nerve. In acute cases the diet should be light, the bowels kept open, and salines may be given. General and local diaphoresis are often useful. Hot fomentations or linseed-meal poultices should be applied to the affected part, or leeches in very severe cases. On the other hand, cold is recommended for traumatic cases. In later stages, counter-irritation by blisters, mustard-plasters, or liniments may be used, if the increased sensitiveness of the skin to these agents be not forgotten (p. 171). Internally, mercury in small doses is the best remedy. For chronic cases, counter-irritation should be employed, and electricity in the form of galvanism. Gowers recommends the anode on the inflamed nerve, or painful spot, and a just perceptible current flowing continuously for ten minutes. Stronger currents or faradism are only desirable in very chronic cases. He advises the muscles to be left alone, unless much degenerated, when they may be stimulated by gentle friction or by a weak interrupted galvanic current.

Multiple Neuritis.

Ætiology.—Multiple or peripheral neuritis (polyneuritis: disseminated neuritis) is the explanation of numerous forms of paralytic affection, formerly thought to have a spinal origin, such as those following chronic poisoning by alcohol and lead. Alcohol, indeed, is its most common cause, and it may also be due to the influence of lead, arsenic, copper, bisulphide of carbon, carbonic oxide, and ergot; it arises in connection with certain infectious or septic diseases, especially diphtheria, but also with simple pharyngitis, scarlatina, rubella, measles, small-pox, typhus, enteric fever, tuberculosis, cerebro-spinal fever, influenza, mumps, cholera, malaria, syphilis, pneumonia, septicæmia, puerperal conditions, rheumatism, gout, and diabetes; and from exposure to cold, damp, or fatigue. Multiple neuritis has already been described as a part of leprosy and of the endemic disease beri-beri. It is probably the explanation of many cases called acute ascending paralysis, or Landry's paralysis.

Its relations to age, sex, or climate, &c., are determined in each case by the disease or agent which has induced it. It will be sufficient here to state that as a result of alcoholism, it is much more frequent in women than in men, and occurs mostly in the middle period of life.

Morbid Anatomy.—The changes in the nerves have been already described—they may be interstitial or parenchymatous. Interstitial lesions are more marked in the larger and medium-sized nerve-trunks, and parenchymatous lesions in the peripheral parts. The changes are slighter as one approaches the spinal cord, and the anterior roots are usually normal. The spinal cord is generally found healthy. The muscles are degenerated or atrophic.

The clinical picture of multiple neuritis varies somewhat according to its origin. One of the most familiar forms is that which follows chronic alcoholism, and the alcoholic paralysis, which will be here described, sufficiently represents what is seen in cases arising from many other causes; but separate descriptions are required for the neuritis of diphtheria (p. 111), of beri-beri (p. 94), and of lead-paralysis (*see* Lead-poisoning).

Symptoms.—These begin generally with tingling, or "pins and needles," in the extremities, or a sensation of numbness; at first only in the hands and feet, later, spreading up the limbs. After a shorter or longer time the limbs become weak. Ross observed that the earliest symptoms are: (1) numbness of the fingers and toes; (2) vasomotor spasm in the fingers and toes, producing "dead fingers"; and (3) muscular cramps, especially in the calves. In acute cases there may be constitutional disturbance, with elevation of the temperature to 103° or 104°; but often the symptoms are not prominent enough to lead to an examination. The patient may, for a time, get about his ordinary occupations, but at last has to take to his bed. The paralysis affects the extensors more than the flexors of the limbs, so that the patient is unable to extend the hand, and the toes are pointed as he lies in bed (drop-wrist and drop-foot). While he can yet walk, he has the high-stepping gait already described under beri-beri (p. 95). The interossei and other muscles are weakened; and, in marked cases, the diaphragm and the vocal cords are paralysed, so that breathing is difficult, and the voice is weakened or abolished. A quick pulse may accompany the laryngeal paralysis, from neuritis of the vagus. The facial muscles may also be affected.

The weakened muscles quickly atrophy; and the change is early noticed in the anterior tibial muscles, in the extensor brevis digitorum, the calf muscles, and the interossei of the hand.

The electrical conditions of the muscles are also altered. The reaction to faradism is diminished or lost, but the effect of the galvanic current varies. Sometimes there are well-marked polar changes, so that complete reaction of degeneration is present. Sometimes there is only a quantitative change.

Sensory symptoms vary considerably—anæsthesia is generally limited to the lower parts of the limbs, but there may be hyperæsthesia. "Pins and needles" or severe gnawing or burning pains belong especially to the early stages. The nerve-trunks are often tender, or their compression may cause "pins and needles" or "deadness"; but the most constant is extreme tenderness of the muscles to pressure, a condition which lasts for months, even in advanced stages of atrophy; it is usually well marked in the muscles of the calf. The reflexes are generally lost. The bladder and rectum are often unaffected, but the excretions are sometimes passed in bed in alcoholic cases, possibly from the accompanying mental state. As the case progresses the skin undergoes trophic changes. Contraction of the limbs may arise, such as flexion at the elbow and pointing of the foot, and adhesions may form in and about the joints. Bed-sores are less common than in spinal cases. With extreme muscular wasting of the body or limbs, it is often remarkable how the form of the face is preserved. The paralysis is generally accompanied, and sometimes partly masked, by a peculiar condition of mind, which is most frequent in alcoholic neuritis, but has been observed by Korsakoff in multiple neuritis from other toxic causes (puerperal fever, typhoid). He therefore calls it *Psychosis polyneuritica* or *Cerebropathia psychica toxæmica.* The essential features are loss of memory for recent events, and delusions as to events that have never happened. Thus, the patient will be unable to tell her name, age, the day of the week, or where she comes from; but, on the other hand, may say that she has been for a walk, or ride, or has seen certain friends, in obvious antagonism to facts. There may be a stage of talkativeness, or even delirium; but, for the most part, the patients show extreme apathy, and complete indifference to surroundings.

Sometimes the symptoms are marked by *inco-ordination* (ataxia), instead of simple paralysis. This may affect the arms or the legs, and may, in the latter case, closely simulate locomotor ataxy of spinal origin.

Diagnosis.—Extensor paralysis of the arms and legs, with wasting and tenderness of the muscles, is characteristic of most cases. In all the combination of sensory symptoms with muscular atrophy and paralysis in all four limbs, especially if the face be involved as well, helps the diagnosis from the spinal paralysis due to disease of the anterior cornua. The ataxic cases are to be distinguished from locomotor ataxy by tenderness of the calf-muscles, by a gait in which the dropping of the foot is noticeable, whereas the foot is often thrown up in tabes, and by the absence of Argyll-Robertson pupil.

Prognosis.—Alcoholic cases may be fatal, either in an early stage, where the cause is not promptly removed; or after months, from emaciation and bed-sores, or phthisis, or cirrhosis of the liver. In many cases the symptoms develop rapidly in the first six or eight weeks, and then the condition of the patient may remain stationary for months, or very slowly improve. After years recovery may be only partial. But in alcoholic and in other cases, where the disease is not too far advanced, and the cause can be completely removed, the prognosis is more or less favourable, recovery taking place slowly in the course of two to six months.

Treatment.—The patient should have complete rest on a water-bed, if the disease is extensive, and nutritious diet, with entire abstinence from alcohol, in cases due to drink. In those due to syphilis, sodium iodide and mercury should be given, and sodium salicylate is recommended for cases following cold. Subcutaneous injections of strychnine have been used with advantage, the doses employed being from $\frac{1}{80}$ to $\frac{1}{20}$ grain two or three times a day. Pains may be relieved by Indian hemp, belladonna, or morphia; by the application of chloroform locally, or by wrapping the limb in cotton wool. Galvanism to the muscles by a continuous current, slowly interrupted, may be used, and massage, if the patient can bear it. Care must be taken to prevent contraction of the limbs.

NEUROMA.

Growths in the nerves may consist of nerve tissue (true neuroma), or of the same tissues that form tumours in other parts (false neuroma). The former consists of medullated or non-medullated fibres, with varying amounts of connective tissue between the fibres; multiple neuromata commonly consist of this variety. On the other hand, a false neuroma is mostly single, and consists of sarcoma, myxoma, carcinoma, syphilitic gumma, especially in the cranial nerves, glioma very rarely, and fibroma most commonly of all. The subcutaneous ends of the sensory nerves are sometimes enlarged into minute tumours, which are visible as small nodules, and may be very painful (cutaneous neuromata, tubercula dolorosa). Another form is *plexiform neuroma*, in which nodular, tortuous, interlacing cords are mixed up with much connective tissue. This form commonly begins in fœtal life; and multiple neuromata are sometimes hereditary, and are said to be associated with a neurotic disposition. Injury from wounds and punctures causes neuromata, as in the case of the painful bulbous ends which form after amputation.

The **Symptoms** are pain, anæsthesia, numbness, and formication in the distribution of the nerve, and paralysis of muscles supplied by it, or more commonly reflex spasms in adjacent or even distant muscles. The tumour may be sometimes felt.

Treatment by medicine is only likely to be successful in syphilitic cases; otherwise the tumours must be removed.

LESIONS OF CRANIAL NERVES.

OLFACTORY NERVE.

A diminution or loss of the sense of smell (*anosmia*) arises from altered conditions of the nasal mucous membrane, such as excessive dryness, or coryza; and in affections of the base of the skull, involving the olfactory bulbs, such as injury, tumours, caries of the bone, and meningitis. It sometimes occurs in locomotor ataxy, and is not uncommon in hysteria, as a part of hysterical hemianæsthesia. It has sometimes occurred after excessive stimulation of the olfactory nerve by strong odours. It should be remembered that loss of smell may affect the power of appreciating flavours, which really requires the combined action of the sense of taste and the sense of smell through the posterior nares.

Excessive sensibility to odours (*hyperosmia*) is noticed in hysteria and insanity; and morbid subjective sensations occur in the insane, and sometimes as an aura in epilepsy.

The primary cause of these defects must be treated if possible. In anosmia galvanism may be tried, the positive pole to the mastoid, the negative to the nasal bones. Strümpell recommends painting with a one per cent. solution of strychnine nitrate in olive oil.

OPTIC NERVE.

The optic nerve has to be considered in connection with its posterior continuations into the brain, the optic chiasma, and the optic tract, which last can be traced through the optic thalamus to the occipital lobes. The optic nerve is really the equivalent of the posterior nerve-roots and the posterior columns of the spinal cord, the lower visual neuron being contained entirely within the retina.

Optic Nerve.—This is liable to inflammation (optic neuritis) from a variety of causes, some arising in the orbit, others in the intra-cranial cavity, whilst others are general conditions of disease mostly affecting the blood. The causes are: (1) in the orbit—orbital tumours, aneurysms of the ophthalmic artery, and rheumatic inflammation; (2) in the cranial cavity—cerebral, cerebellar, and meningeal tumours of all sizes and in all situations, acute and chronic meningitis, chronic hydrocephalus, and thrombosis of the cerebral sinuses; (3) general causes—infective endocarditis, Bright's disease, leuchæmia, chlorosis, lead-poisoning, enteric fever, and some other conditions. The appearances of optic neuritis and its effects on vision have already been described (p. 176). When it is due to orbital disease, it occurs only on the same side as the affected orbit, and is double only when both orbits are diseased; but intra-cranial lesions, whether unilateral, median, or multiple, as well as blood conditions, cause, with rare exceptions, a *double optic neuritis*, unless the lesion—as, for instance, a carotid aneurysm—is so far forward as to press on the nerve in front of the chiasma.

Retrobulbar neuritis affects the nerve behind the eyeballs, and causes no change visible with the ophthalmoscope. It may give rise to rapid blindness, severe headache, or pain in the eyes, aggravated by movements of the eyeballs on pressure upon them in a backward direction. *Axial neuritis*, or neuritis of the central fibres of the nerve, leads to central loss of vision.

Optic atrophy occurs as a late stage of optic neuritis (secondary atrophy), and occasionally as a primary lesion, without any previous neuritis, as in locomotor ataxy. Vision is very greatly reduced, or abolished entirely, by optic atrophy.

Optic Chiasma.—This may be affected by tubercle or syphilitic gumma or meningitis, by the pressure of internal hydrocephalus, and by hæmorrhage into its substance. Since it contains the decussation of those fibres which pass from the left tract to the nasal half of the right retina, and those which pass from the right tract to the nasal half of the left retina, it follows that its lesions produce paralysis of the *nasal* half of each retina, and a corresponding blindness in the *temporal* half of each visual field. This is accordingly called *temporal hemianopia*. If the lesion extends to one side, or forward in the optic nerve, or backwards in the optic tract, the direct fibres on that side are affected as well, and the vision of that eye is quite lost.

A *nasal hemianopia*, or blindness of the inner half of each field, would result from a separate lesion on each side of the chiasma, involving the direct fibres to the outer half of the retina. It is necessarily very rare.

Optic Tract.—The optic tract contains fibres which pass to the visual centres, partly direct from the outer half of the eye of the same side, partly across the chiasma from the inner half of the retina of the opposite eye. A lesion of the *left* optic tract paralyses the left half of each retina, and gives blindness in the *right* half of each field; similarly, a lesion of the right optic tract produces blindness of the left half of each visual field. This form of blindness is called *lateral* or *homonymous hemianopia.* It may be caused not only by lesions of the tract itself, but by diseases of the brain implicating the fibres which connect the tract with the occipital lobe and the visual centres. The dark part of the field may be a complete half, or it may be less; and this partial hemianopia is more likely to arise from lesions in those posterior situations where the conducting fibres are less closely united together.

A tumour in the occipital lobe may thus cause lateral hemianopia; and if each occipital lobe is successively or simultaneously affected by any lesion there will be complete blindness, or double lateral hemianopia. Optic neuritis may in some such cases be entirely absent, and is not a necessary part of lateral hemianopia. A transient hemianopia may occur in cerebral hæmorrhage; and a peculiar form of lateral hemianopia is a striking phenomenon in migraine. In recovery from hemianopia, the field generally clears from centre to periphery, as is common in migraine, or from above or from below; but rarely from periphery to centre.

A patient with lateral hemianopia is likely to keep his head turned towards the blind side, in order to see distinctly things in front of him. If the left is the blind side, the right half of each retina is paralysed; if then the head is turned to the left, objects in front of the patient fall upon the left, or normal, half of each retina. In lateral hemianopia, colour vision may also be affected (*hemiachromatopia*).

Another form of visual defect is that known as *crossed amblyopia*, or blindness of one eye due to a lesion on the opposite side of the brain. The lesion then must be behind the tract; and it appears probable that this may be caused by some disease of the cortex of the lower and hinder part of the parietal lobe, the supra-marginal, and the angular convolutions. There is concentric reduction of the field of vision for white light; and the colour field is also reduced, and may be lost entirely. If the apparently sound eye is tested, a much less degree of reduction of the field, both for white light and colour, will also be found. Thus it would seem that the visual centre on one side has connections with the eyes of both sides. In some cases of lateral hemianopia, there is concentric reduction of the half-field which is not blind, and this reduction is greatest in the field of the eye opposite to the lesion. It is suggested by Gowers that this is due to an extension of the lesion from the centre corresponding to the lateral hemianopia, to that higher centre whose destruction causes crossed amblyopia.

Crossed amblyopia is frequently seen in hysteria, associated with hemianæsthesia.

THIRD, FOURTH, AND SIXTH NERVES.

These nerves supply the muscles which move the eyeball, and their lesions are best considered together. The fourth supplies the superior oblique muscle, the sixth the external rectus, and the third the superior rectus, inferior rectus, internal rectus, and inferior oblique, as well as the ciliary muscle, the sphincter of the iris and the levator palpebræ muscle. The constant association of the eyes together in all their movements leads, when any one muscle is paralysed, to some important motor and visual disorders, by which the paralysis may be recognised. These are:—limited movement, strabismus, secondary deviation of the sound eye, erroneous projection, and diplopia.

Limitation of Movement.—This is in proportion to the amount of paralysis: in extreme cases—for instance, of paralysis of the external rectus—the eye cannot be moved outwards beyond the middle line. After a time contracture of the internal rectus takes place, and the eyeball is turned into the inner canthus.

Strabismus, or squinting, is a want of correspondence of the visual axes. This occurs when the two eyes look at an object which is in the part of the field corresponding to the action of the paralysed muscle. Thus, if the right external rectus is paralysed the right eyeball cannot move outwards; and when an object in the right half of the field is looked at, the left eyeball moves to the right, while the right eyeball remains stationary; the visual axes converge, and so much the more strongly the farther to the right the object is situate. This is called *convergent* strabismus. If the right internal rectus is paralysed, the left eye can follow an object to the left, but the right remains stationary, looking forwards. The visual axes diverge, and so much the more strongly the farther the object is to the left. This is *divergent* strabismus. The divergence of the axis of the affected eye from correspondence with that of the sound eye is called the *primary deviation*.

Secondary Deviation.—If, under the above circumstances, while the sound eye is looking straight at (or fixing) the object, it be covered, and the affected eye be made to fix the object, the sound eye will be moved still farther in the same direction—that is, inwards with paralysis of the opposite external rectus, outwards with paralysis of the opposite internal rectus. This is called secondary deviation. It may be further measured if, subsequently, the affected eye be covered, and the sound eye, again fixing the object, returns to the position it formerly occupied. Secondary deviation is the result of the increased innervation thrown into the paralysed muscle, and acting with the greater effect upon the unparalysed muscle opposite.

Erroneous Projection.—We judge of the position of objects in relation to our own bodies by the movement of the eyeball, or rather by the amount of nerve force supplied to the muscles that move it. If the eyes are at rest in the middle of the orbit, we know that an object in the middle of the field is straight in front of the body; if it moves, and we follow it with the eyes, we judge of its new positions by the movement of the eyeballs. If a muscle is paralysed the increased effort to move suggests a greater movement than has really taken place, and so gives the idea that the object is farther in the direction of the movement attempted than it really is. If the patient tries to touch it with his finger he strikes too far in that direction and may miss it entirely. The erroneous projection is always in the direction of the action of the paralysed muscle: outwards, in paralysis of an external rectus; inwards, in paralysis of an internal rectus; upwards, in paralysis of a superior rectus.

Diplopia, or Double Vision.—In paralysis of an ocular muscle there is separate vision with each eye, when the visual axes do not correspond. The *true* image seen by the sound eye, which fixes, is clear and sharp; the *false* image, seen by the inactive eye, is less distinct, because it does not, like the other, fall upon the yellow spot. There are two forms of diplopia: *lateral* or *homonymous diplopia*, in which the right-hand image corresponds to the right eye, and the left-hand image to the left eye; and *crossed diplopia*, in which the right-hand image belongs to the left eye, and the left-hand image belongs to the right eye. Lateral diplopia occurs with convergent strabismus, crossed diplopia with divergent strabismus. In paralysis of the lateral muscles during horizontal movements the images are parallel with one another on the same level; but in other movements and with other paralyses they are on different levels, or inclined to one another.

Diplopia has been generally explained by the fact that the image of the object forms on parts of the two retinæ that do not correspond. Gowers prefers to explain it by the erroneous projection of the image seen by the affected eye in the direction of the action of the paralysed muscle.

In examining cases of diplopia a piece of coloured glass may be placed over one eye to distinguish the image that corresponds to it.

Diplopia may be diminished or increased by the use of prisms: diminished if the prism is placed with its base in the direction of action of the paralysed muscles; increased if it be placed in the other direction.

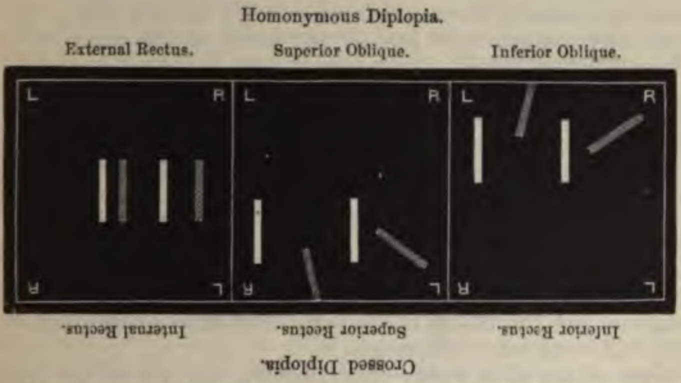
Muscle Paralysed.	Diplopia.	Half of Field.	Relative Position of Images to one another.	Effect of Movements on Relative Position of Images when looking—	Defect of Movement.	Secondary Deviation, by—	Strabismus on Movement in Direction of Deficiency (column 6).	To avoid Diplopia, Face turned towards—
External rectus.	Homonymous.	Outer.	Parallel and at same height.	Outwards—separation increases.	Outwards.	Opposite internal rectus.	Convergent.	Affected side.
Superior oblique.	Ditto.	Lower.	False lower than true; inclined to it at upper end; appears to be *nearer*.	Downwards and outwards—difference of height is less, inclination is greater. Downwards and inwards—*vice versâ*.	Downwards and outwards.	Opposite superior oblique.	Convergent only below horizontal plane.	Sound side, head down.
Inferior oblique.	Ditto.	Upper.	False higher than true; inclined to it at lower end.	Upwards and outwards—difference of height is less, inclination is greater. Upwards and inwards—*vice versâ*.	Upwards and outwards.	Opposite inferior oblique.	Convergent only above horizontal plane.	Sound side, head carried high.
Internal rectus.	Crossed.	Inner.	Parallel and at same height.	Inwards—separation increases.	Inwards.	Opposite external rectus.	Divergent.	Sound side.
Superior rectus.	Ditto.	Upper.	False higher than true; slightly inclined to it at lower end.	Upwards and outwards—difference of height is greater, inclination is less. Upwards and inwards—*vice versâ*.	Upwards.	Opposite superior rectus.	Eyeball rotated upper end outward.	Affected side, head carried high.
Inferior rectus.	Ditto.	Lower.	False lower than true; slightly inclined to it at upper end.	Downwards and outwards—difference of height is greater, inclination is less. Downwards and inwards—*vice versâ*.	Downwards.	Opposite inferior rectus.	Eyeball rotated upper end inward.	Affected side, head down.

The results of paralysis of the individual muscles are given on p. 188, in a tabular form, and should be compared with Fig. 10.

Fourth and Sixth Nerves alone.

Lesion of the fourth nerve causes paralysis of the superior oblique muscle, with the results shown in the table: this nerve is very rarely diseased alone. Lesion of the sixth nerve causes paralysis of the external rectus.

Fig. 10.



Images as seen in Diplopia from Isolated Paralysis of Ocular Muscles of Right Eye. The Patient is supposed to be in the position of the Reader. The True Image is White, the False Image Shaded. Two Positions in the Field are given for each form of Paralysis.

Third Nerve alone.

This nerve has much wider connections. Its lesions cause paralysis of the superior rectus, inferior rectus, internal rectus, inferior oblique, levator palpebræ, sphincter of the iris, and ciliary muscle. In complete paralysis the eye can only be moved outwards by the external rectus, and downwards and outwards by the superior oblique; at the same time there is *ptosis* or drooping of one upper eyelid from paralysis of the levator palpebræ. There is diplopia in the upper, inner, and lower parts of the field, the false image higher than the true, and the separation of the images increases with movement inwards. The strabismus is divergent. The eyeball is slightly prominent from the weakness of the recti, and in late cases there is contracture of the external rectus. Accommodation is paralysed, and the pupil is dilated.

Lesions of the third nerve are, however, often partial, and accordingly one or more muscles may escape—for instance, ptosis may be absent; or the levator palpebræ and superior rectus, supplied by the upper division of the nerve, may be affected alone or escape together; or the external muscles may be alone affected, or the internal (ciliary muscle and pupil contractors) alone.

Internal Ocular Paralysis.—*Cycloplegia* is the loss of the power of accommodation from paralysis of the ciliary muscle. Distant vision is clear, but near vision is blurred and indistinct. It may occur alone, or there may be at the same time an absence of contraction of the pupil, which takes place normally with accommodation. The ciliary muscle is supplied by the third nerve by means of fibres which arise in the anterior part of the third nerve-nucleus, and pass by the trunk of the nerve to the ciliary ganglion and the ciliary nerves.

Iridoplegia.—Paralysis of the iris occurs in three forms.

Accommodative iridoplegia.—This is a want of contraction during accommodation for near vision.

Reflex iridoplegia.—Loss of pupil light-reflex, or absence of contraction of the pupil to the stimulus of light. The reflex path is through the optic nerve, both optic tracts, probably the corpora quadrigemina, the anterior part of the nucleus of the third nerve behind the centre for accommodation, probably the second fasciculus of the origin of the third nerve, its trunk, the ciliary ganglion, and the ciliary nerves; and any sufficient lesion of this path will cause reflex iridoplegia.

Loss of the skin reflex.—On pinching the side of the neck, the pupil of the same side will be noticed to dilate, and the same may happen when the palm of the hand is tickled. The motor path of this reflex is in the cervical sympathetic, and the fibres connecting this with the cord at the lowest part of the cervical region. The centre is said to be beneath the corpora quadrigemina. In some diseases involving this path the reflex is absent.

Ophthalmoplegia.—By this term is meant paralysis of the ocular muscles in general. *External ophthalmoplegia* is paralysis of the muscles outside the eyeball; *internal ophthalmoplegia*, paralysis of the pupil and ciliary muscle; *total ophthalmoplegia* is paralysis of both internal and external muscles.

The lesions causing ocular paralysis may affect (1) the trunks of the nerves: these are syphilitic and so-called rheumatic inflammations, the pressure of orbital or intra-cranial growths, or of aneurysms, and rarely tumours of the nerves themselves; (2) the fibres in the brain connecting the nerves with their nuclei: such as hæmorrhage, softening, tumours, and disseminated sclerosis; (3) the nerve-nuclei. Paralyses of ocular nerves occur in connection with some diseases, without our being able to say with certainty what is the seat or the nature of the lesion.

Diphtheritic paralysis often begins with strabismus and loss of accommodation. In locomotor ataxy paralysis of the third, fourth, or sixth nerve occurs, which may be permanent or transient. A pupil contracting with accommodation but not to light (*Argyll-Robertson pupil*) is very common, and one of the earliest symptoms; sometimes it is closely contracted. Syphilis may act by causing meningitis or gummatous thickening, and may contribute to the formation of aneurysms. Nuclear degenerations also occur with some frequency in syphilitic subjects. Cases of *relapsing* or *recurring paralysis* have been described. They begin with pain in the eye, often with headache and vomiting. These last two or three days, and coincidently, or as they lessen, there is paralysis of several ocular muscles, internal and external. Ptosis is generally present. The paralysis lasts a few days or weeks, and recurs at intervals of months or a year. The disease lasts from early life to middle age. The cause is obscure.

Ocular Paralysis from Disease of the Nuclei.—Ophthalmoplegia is a common result of disease of the nuclei. Some cases with an acute origin have been recorded, but chronic ophthalmoplegia is much more frequent. It occurs in association with locomotor ataxy, and in syphilitic subjects; and also with progressive muscular atrophy, bulbar paralysis, and general paralysis of the insane; it is gradual in its course, first one or two muscles, then others, and finally, after some years, nearly all the muscles, being affected. Ptosis, however, may be absent, and is rarely complete. If present, it gives the patient a sleepy look; the eyeballs are fixed or staring, and sometimes they are slightly prominent. Double vision may be present in the early stages, but often disappears in the course of time. Degenerative changes were found in the ocular nerve-nuclei by Gowers in a case of Hutchinson's. A nuclear lesion is certainly present if the internal muscles are alone paralysed in both eyes, or if the external muscles are paralysed in both eyes without the internal, or if associated muscles in the two eyes are paralysed at the same time. But if both external and internal muscles are paralysed in both eyes, coarse disease of the base of the brain may be a cause, though nuclear lesions are far more probable.

A lesion on one side of the pons affecting the nucleus of the sixth nerve causes *conjugate deviation* of the eyes to the opposite side. The external rectus is paralysed directly, the internal rectus of the opposite eye is paralysed through commissural fibres between the sixth nerve-nucleus and the opposite third nerve-nucleus, and through the fibres to the internal rectus arising there. A lesion of the sixth nerve-nucleus is often accompanied by some facial paralysis from the close proximity of the facial nerve-fibres which wind round it.

Ptosis.—Besides ptosis due to lesions of the fibres and nucleus of the third nerve, there are other forms :—*reflex ptosis*, from irritation of the third nerve, especially by decayed teeth; ptosis from paralysis of the fibres of Müller in the fascia of the orbit, which are supplied by the sympathetic; *congenital ptosis*, probably due to a central defect; ptosis in weakly adults, especially women, which is worse in the morning; and, finally, *hysterical ptosis*. This last affects both sides; the head is thrown back, and the frontales may be strongly contracted when the patient tries to look up. But the attempt is accompanied by a spasmodic movement of the orbicularis, which prevents the action of the levator.

Treatment of Ocular Paralysis.—This must depend upon the cause, if it can be ascertained. Syphilitic cases should be treated by potassium iodide and mercury, and the same drugs may be at least experimentally given in many other cases of less certain origin. For cases of an inflammatory nature, counter-irritation by a blister behind the ear or leeches to the temple should be tried. If cold has appeared to be the cause, hot fomentations should be applied, and salicylates might be given internally. Galvanism may be applied by holding the well-wetted rheophore in one hand and applying the forefinger of that hand to the closed eyelid, opposite the paralysed muscle; the other rheophore is on the patient's neck (Buzzard). Diplopia may be relieved by the use of a prism, which should not be strong enough to fuse the images, but only to approximate them, so that muscular efforts may be encouraged. For ptosis in weakly individuals, tonics, such as iron, quinine, and strychnia, are required, as well as local stimulants. Similar remedies, and especially stronger stimulants, such as blisters to the temples, and faradism, should be used in the hysterical form; if one eye only is affected, the other eye should be bound up.

Fifth Nerve.

The fifth, or trigeminal nerve, has both sensory and motor functions, represented by a large sensory, and a small motor root. The sensory root enters the Gasserian ganglion, and in front of it divides into its first, second, and third divisions; the third division is joined by the motor root. The first division receives a branch from the sympathetic, which goes to the dilating fibres of the iris; the second division is connected with the spheno-palatine ganglion; the third with the otic ganglion and the chorda tympani nerve. The third division is called lingual gustatory, but it is probable that all taste-fibres pass into the chorda tympani, by which they reach the facial nerve, pass along it to the genu, and, leaving it by the great superficial petrosal and vidian, pass through the spheno-palatine ganglion to the second division of the fifth.

The fibres of the fifth nerve may be injured in any part of its course. In the pons Varolii its origin may suffer from tumours or hæmorrhage; its trunk may be affected by tumours or meningitis at the base of the brain; in front of the Gasserian ganglion the first division is liable to pressure from tumours about the cavernous sinus, or aneurysms or cellulitis in the orbit; the second and third divisions may be injured by growths in the sphenopalatine fissure. Injuries to the mouth or nose may involve various branches of the second and third divisions, and neuritis of the same may be caused by neighbouring inflammations.

Symptoms.—These must depend on the position of the lesion, and whether it involves the fibres of sensation, taste, or motion. If the *sensory* fibres are involved, the result is anæsthesia of the face, corresponding to the distribution of the nerve; for the first division, anæsthesia of the forehead and anterior part of the scalp, the upper eyelid, and bridge and tip of the nose; for the second division, the malar bone, the cheek, the lower eyelid, the side of the nose, the upper lip, upper teeth, upper part of the pharynx, tonsils, soft palate, uvula, and roof of the mouth; for the third division, the greater part of the temple, the upper and front part of the ear, the auditory meatus, the lower part of the cheek near the mouth, the lower lip, the chin, the lower teeth and gums, the tongue, part of the mucous membrane of the mouth, and the salivary glands.

The loss of sensation is often preceded by tingling and numbness, or neuralgic pains, and there may be tender points like those found in ordinary neuralgia; it may be accompanied at first by sensitiveness to pain, but this is finally lost also. The conjunctiva, and the nasal and buccal mucous membranes, are, of course, involved as well as the skin. The nose is insensitive to the stimulus of pungent vapours like ammonia, and though smells are at first perceived well, the sense becomes afterwards blunted. In consequence of the mouth being insensitive on one side, food is not chewed on that side, and a thick fur collects on the tongue for want of the cleaning operation of mastication.

Certain *trophic* changes also occur in lesions of the fifth nerve; the secretions of the mucous membranes are diminished, there may be swelling and ulceration of the gums, and the teeth become loose; if the cheek is bitten it heals slowly. Often the eyeball inflames (*neuro-paralytic ophthalmia*); keratitis begins on the lower side, with cloudiness, opacity, and ulceration, by which eventually the eye may become perforated and destroyed. It has been attributed to irritation of the insensitive surface by foreign bodies. Gowers suggests that there is a special irritating lesion of the nerve itself. Herpes zoster occurs especially in connection with the first division (*H. z. ophthalmicus*), and appears to arise from inflammation of the Gasserian ganglion.

Loss of taste occurs in disease of the root of the nerve, and may then be complete. Loss of taste in the anterior two-thirds of the tongue occurs from lesions of the lingual gustatory *below* the junction of the chorda tympani. To explain the complete loss in lesion of the root, we must believe that the taste fibres from the back of the tongue reach the fifth nerve by some circuitous course, like those of the anterior two-thirds. Such a course may be through the glosso-pharyngeal nerve, by its tympanic branch to the petrosal nerve and otic ganglion. Gowers notes the facts that there is no case on record showing abolition of taste from disease of the root of the glosso-pharyngeal nerve, and that taste is often lost in the back as well as the front of the tongue from caries of the middle ear.

If the *motor* portion of the fifth nerve is involved, which is only likely to happen in lesions near the origin of the nerve, the temporal, masseter, and pterygoid muscles are paralysed. In the first two, this can be detected by placing the hand on the temple or the side of the jaw while the teeth are firmly clenched, when a comparison with the other side will detect the want of contraction. If one external pterygoid is paralysed, the jaw cannot be moved to the opposite side; and if the jaw is depressed it deviates to the paralysed side. Any impairment of the action of the mylohyoid and digastric, and of the tensor tympani and tensor palati, cannot be detected. After a time atrophy of the temporal and masseter muscles may be recognised.

Diagnosis.—The presence of severe pain may give for a time a resemblance to neuralgia; but anæsthesia and loss of taste prove an organic origin. If one or other branch is alone affected, the lesion is in front of the Gasserian ganglion; if all the branches, it must be near the origin. The association of other nerve-paralyses, such as those of the ocular nerves, or of the motor tract, may also help to localise. Loss of taste occurs as a part of hemianæsthesia, and of hysteria.

Treatment.—Besides dealing with the cause, if possible, we may relieve pain by morphia, cocain, or gelsemium; and anæsthesia may be treated by the application of the faradic wire-brush.

SEVENTH OR FACIAL NERVE.

The tortuous course of the facial nerve from the pons to its distribution on the face renders it especially liable to inflammation and compression. Paralysis of the facial muscles, indeed, may be caused by lesions not only of the facial nerve itself, and of the nerve-nucleus, but also of the facial portion of the cortical centres, and of the fibres which connect this with the nucleus. These last, supra-nuclear, lesions cause a limited form of paralysis, which will be described in *hemiplegia*. We have here to do with the more complete nuclear and nerve-trunk paralysis which goes sometimes by the name of *Bell's palsy*.

Causes.—The nucleus and the fibres in the pons may be involved in tumours in that part of the brain, and occasionally the nucleus is degenerated as a part of *labio-glossal paralysis*. Tumours of all kinds at the base of the brain and meningitis may involve the nerve-trunk between the brain and the internal auditory meatus. In the petrous bone the nerve is liable to injury from otitis, and suppuration of the mastoid cells; and rarely hæmorrhage has compressed the nerve in the aqueduct of Fallopius. On the side of the face the nerve may be injured by blows, or may be involved in cellulitis or parotid growths. The more common cases of facial paralysis often come on after exposure of the face to a draught, as by sitting at a window; but sometimes without any recognisable cause. Such cases are attributed to neuritis, which probably affects the nerve at its emergence from the stylo-mastoid foramen. A few cases of facial paralysis have been recorded after zona of the face, when it must be supposed that the neuritis first affecting the branches of the sensory fifth nerve has spread to those of the motor seventh. Facial paralysis is nearly always unilateral. Double facial paralysis (*diplegia facialis*) may occur from bilateral disease of the pons, or from otitis, or syphilitic disease successively affecting both nerves. Diphtheria, syphilis in the secondary stage, and influenza appear also to cause double facial neuritis. An incomplete, yet double, facial paralysis may occur as a part of multiple neuritis.

Symptoms.—In typical cases the muscles of the affected side of the face are more or less paralysed. The most noticeable features are the inability to close the eye, and the distortion of the mouth on attempting to smile or show the teeth; but other defects may be seen on examination. The forehead is not wrinkled on trying to raise the eyelids or look up. The eyelids cannot be brought together, but there is a permanent fissure of a quarter to a third of an inch in width (*lagophthalmos*). When told to close the eye, the patient brings the lids nearly together, and then rolls the eyeball under the upper lid, so that only the sclerotic can be seen. On smiling or showing the teeth, the angle of the mouth is drawn up on the healthy side; but the lips remain in contact on the paralysed side, and a characteristic elongated triangular opening is the result. Moreover, the lips, and the median fossa under the nose, are displaced to the sound side, so that the tongue, if protruded, *seems* to lie nearer the paralysed side, though actually in the middle line. This can be proved by looking at the incisor teeth. The lips cannot be put together for whistling, or blowing, and air escapes irregularly on the paralysed side; the articulation of labial sounds is imperfect in bad cases. The buccinator is paralysed, and food collects between the cheek and the gums. The failure of the nasal muscles may be seen in efforts to sniff, when the nostril on the sound side dilates, while the opposite ala remains passive. The paralysis of the depressors of the angle of the mouth and of the platysma can also be shown. An important difference exists between the condition in youth and advanced life. The elasticity of the tissues in youth will keep the different parts of the paralysed face in their normal position so perfectly, that during rest there is no want of symmetry, and the paralysis may be quite overlooked until the patient speaks or smiles; but in advancing age elasticity is lost, wrinkles multiply, and the parts which are unsupported by muscular action fall by the action of gravity, and cause a distortion which can be at once recognised. The lower lid falls away from the eyeball, the lachrymal secretion may thence overflow on to the cheek, and the lower angle of the mouth is depressed. The levator palati, formerly said to be innervated by the facial nerve and the spheno-palatine ganglion, is probably supplied by the spinal accessory through the vagus: it is not affected in facial paralysis. Taste may be lost on the affected side of the tongue, if the facial is diseased in its bony canal between the genu and origin of the chorda tympani nerve. There may be increased sensitiveness to musical notes of low pitch, from paralysis of the stapedius and unopposed action of the tensor tympani. Deafness may be present either from co-existing lesions of the auditory nerve or from aural catarrh.

The electrical reactions undergo changes similar to those in other muscles paralysed by lesions of nerve and nucleus. Faradic reactions are diminished or lost; and galvanic reactions, at first excessive, subsequently become diminished with the development of polar changes.

If wasting occurs in late stages it is not obvious, as the muscles are so thin as to contribute very little to the natural fulness of the face.

Course.—Facial paralysis runs a variable course. The so-called rheumatic form often develops quite suddenly, and is complete in a few hours. It may gradually recover in a few weeks or months; it may recover only partially; or the face may remain permanently and absolutely paralysed. A partial recovery is often followed by a peculiar condition of contracture of the paralysed muscles. These are somewhat shortened, the eye is a little closed, and the angle of the mouth is slightly drawn up by the zygomatici; and, if the muscles of the sound side are at rest, the first impression that one gets is that the paralysed side is active, and that the sound side is paralysed. This idea is corrected at once when the patient speaks, or smiles, or tries to shut the eyes. The contracted side can contract very little more, while the sound side has a wide range of movement. In this condition, also, the affected muscles cannot be moved independently—in closing the eye, the angle of the mouth is raised; in smiling, the eye is partially closed. This is called *secondary over-action.*

Diagnosis.—The recognition of facial paralysis is not difficult. The important point is generally to distinguish the seat of the lesion. In facial paralysis of cerebral or supra-nuclear origin, the muscles of the lower part of the face are mostly affected, those of the upper part very little; the eye can always be closed, though not so tightly as on the opposite side; and the wrinkling of the forehead is slight. A smile is less impaired when produced by emotion than when voluntarily attempted, and the electrical reactions of the affected muscles are normal, or nearly so. In a lesion of the facial nerve-nucleus, the orbicularis oris escapes, as the nuclear origin of the nerve of this muscle seems to be connected with that of the tongue. A tumour of the pons not infrequently involves the seventh and sixth nerves at the same time from their close proximity to one another. Lower down the loss of taste may localise the lesion as indicated. Rheumatic neuritis may begin below this section of the nerve, and involve it by extension.

Treatment.—Cases of tumour may not be amenable to treatment. Potassium iodide should be given in cases possibly syphilitic. In ordinary rheumatic paralysis, warmth locally applied, counter-irritation by a blister over the mastoid process, and salines with potassium iodide should be employed. Later the continuous galvanic current should be used.

Eighth or Auditory Nerve.

Various cerebral lesions may involve the nuclei of the nerve in the pons, or its higher connections in the brain; the nerve itself may be injured by meningitis, by thickening of the petrous bone, by aneurysms or tumours: lastly, the expansion of the nerve in the labyrinth may be damaged by acute or chronic inflammation, by syphilitic disease, or degenerative changes.

The results of these lesions are; *Deafness;* and various subjective sounds, especially *tinnitus.* Excessive sensibility to sounds is more often a functional disorder.

Deafness from the above cause is called nervous deafness, and has to be distinguished from loss of hearing due to interference with the conduction of sound through the tympanum and external meatus. The examination of the ear for this purpose can be made with the tuning-fork, which should be first held near the ear, and then placed with its foot against the temporal bone. If the sound cannot be heard in the first position, but can in the second, then the nerve apparatus is healthy, but conduction is bad. A healthy nerve apparatus is also shown if with the tuning-fork on the bone the sound is intensified by closing the ear. If the sound cannot be heard with the tuning-fork on the bone, the nerve apparatus is faulty.

Normally, if a vibrating tuning-fork be held to the head till it is no longer heard, and then held opposite the ear, its vibrations are again heard for a time. This fact may be utilised in slighter cases of deafness; if the tuning-fork is *not* heard opposite the ear longer than it is on the bone, the conduction of the ear is impaired.

When the conduction is found to be normal, deafness must be due to a lesion either of the nerve or of the labyrinth. Which of these is more likely to be at fault must be determined by associated symptoms. These may sometimes point to an intracranial lesion, but, as a fact, deafness is not a very common symptom in cerebral cases, unless the trunk of the auditory nerve is directly compressed by a tumour.

Tinnitus Aurium.—This term includes the various subjective sensations of sound, generally of ringing, rushing, or roaring kind, with which, in their slighter degrees, nearly every one is familiar. It is clearly due to irritation of the auditory nerve-fibres, and may occur in almost any form of ear disease, whether of the external meatus, of the middle ear, of the labyrinth, or of the nerve or nerve-centres. An instance of lesion in the first situation is the accumulation of cerumen; instances of lesions in the last situation are subjective sounds experienced in migraine, in epilepsy, and in conditions of impaired general health, such as gout, anæmia, alcoholism, and chronic Bright's disease. Tinnitus is frequently associated with deafness, the two symptoms being due to a common cause.

Treatment.—The cause of the tinnitus must be first considered. Disease of the external or middle ear may be directly treated, and any general disorder, like anæmia or alcoholism, should be met by appropriate remedies. In addition, when the tinnitus seems to be due to the nerve or labyrinth, the following may be tried: Potassium bromide, hydrobromic acid, quinine, sodium salicylate (Gowers) internally; counter-irritation by a blister behind the ear, or the continuous current of electricity.

NINTH OR GLOSSO-PHARYNGEAL NERVE.

There is considerable doubt as to the exact anatomy of this nerve. Its nucleus contains cell-bodies like those of motor neurons; and the petrous ganglion contains the cell-bodies of its sensory neurons, and corresponds to the ganglia of the spinal nerves. It has been already stated that lesions of the fifth nerve cause loss of taste at the back of the tongue, and that no lesion of the glosso-pharyngeal root has been known to abolish taste in this situation. Taste-fibres, if they exist in the nerve, may reach the fifth nerve through the tympanic plexus and the otic ganglion. The nerve is rarely if ever affected alone: the nucleus of the nerve is involved in cases of labio-glossal paralysis, and the trunk is likely to be affected by tumours or other lesions of the medulla, in association with the root of the pneumogastric; so that symptoms referable to disease of the glosso-pharyngeal alone cannot generally be recognised.

TENTH, PNEUMOGASTRIC, OR VAGUS NERVE.

This nerve has both motor and sensory fibres, the latter being connected with the ganglia at the base of the skull: while many of its motor fibres are contributed by the accessory portion of the spinal accessory nerve. The vagus is extensively distributed to the pharynx, larynx, lungs, heart, stomach, intestines, and spleen.

It is liable to still more lesions than the other cranial nerves, from its great extent and varied course—*e.g.*, lesions of its nuclei from degeneration, softening, or hæmorrhage, generally in association with the adjacent nuclei; meningitis, syphilis, tumours, or aneurysm affecting its roots; in the neck wounds, surgical operations, aneurysms, new growths, or enlarged glands. The last three are frequently causes of difficulty with the pneumogastric in the thorax and the recurrent laryngeals in any part of their course. Diphtheritic paralysis and alcoholic neuritis also affect the functions of the pneumogastric nerve.

If the *pharyngeal* branches are affected, swallowing is difficult, the food lodges in the pharynx, and small portions or liquids may pass into the larynx and cause choking. If the *laryngeal* branches are diseased, either in the trunk of the nerve itself, or in the recurrent laryngeal nerve, the various forms of paralysis of the vocal cords, and other parts of the larynx, are produced, which are described in the section on Diseases of the Larynx. *Pulmonary* branches are both afferent and efferent; of the afferent fibres, some stimulate, others inhibit, the respiratory centre, and the efferent fibres are said to supply the muscular fibres of the bronchi. But it is only in rare instances that the results of lesions of these fibres are observed clinically. The phenomenon called Cheyne-Stokes respiration, and the spasms of hydrophobia, are probably dependent on changes in the respiratory centres, with which the vagus nucleus must be connected. The *cardiac* fibres have an inhibitory action, and are believed to be inflamed in cases of alcoholic paralysis, when the pulse may become excessively rapid; a similar acceleration has occurred from local disease of the nerve-trunk. Some curious cases are on record of slowing of the heart from irritation of the vagus by pressure. Lesions of the *gastric* branches seem to have caused in different cases pain, vomiting, or excessive appetite; the vomiting frequently observed in cerebral disease must be due to irritation of these nerves.

Treatment must be conducted on the lines indicated in the case of other nerves. (*See also* Diseases of the Larynx.)

ELEVENTH OR SPINAL ACCESSORY NERVE.

The external portion of this nerve arises by a series of roots from the cervical part of the spinal cord, and is really a motor spinal nerve directly connected with the anterior cornua. It is distributed to the sterno-mastoid and trapezius muscles; it is the chief supply of the former, but the latter is largely innervated by cervical and dorsal nerves.

In addition to cerebral and intracranial lesions, like those suffered by the vagus, the spinal accessory may be injured by caries of the cervical spine, enlarged glands, or abscesses in the neck, or by direct injury. If the lesion is in the posterior triangle, the sterno-mastoid will, of course, be spared. Paralysis of the sterno-mastoid is shown by the want of prominence due to the contraction of the muscles, and by deficient power of rotation of the head to the opposite side. In paralysis of the trapezius, the natural slope between the neck and the shoulder is converted into a deep hollow, which is exaggerated when the shoulder is raised, as it still can be by the action of the levator anguli scapulæ. The point of the shoulder lies lower than normal, and the angle of the scapula is rotated inwards by the unopposed action of the rhomboids and levator. Elevation of the hand above the head is, however, difficult or impossible, because the trapezius does not fix the scapula for the use of the deltoid, nor does it assist in that rotation, for which the serratus magnus is chiefly employed. If the whole muscle is paralysed, the approximation of the shoulder-blade to the spine is incomplete; but in spinal accessory lesions it is chiefly the upper part between the occiput and the acromion which is affected. With a persisting lesion atrophy and electrical changes naturally follow.

Treatment.—Here we must deal, when possible, with the causative lesion, and with the muscular failure by galvanic stimulation, and perhaps massage.

TWELFTH OR HYPOGLOSSAL NERVE.

This nerve, like the last, has a purely motor function, supplying the tongue and most of the muscles attached to the hyoid bone. Its lesions are very similar to those of the two nerves last considered. As the two nuclei are so close to the middle line, they are generally affected together, producing bilateral results.

Unilateral paralysis may result from disease above the nucleus, between it and the cortical centre in the ascending frontal convolution; and below the nucleus, from meningitis, simple and syphilitic growths, caries of the cervical vertebræ, and tumours, cellulitis, or injuries beneath the jaw. If it is paralysed on one side, the back of the tongue on that side is slightly raised, from loss of the tonic contraction of the hyoglossus muscle. In the mouth it cannot be moved freely to the same side, but when protruded is pushed to the affected side by the contraction of the posterior fibres of the genio-hyoglossus, and by the elongating action of the transversus muscle on the healthy side. In bilateral paralysis, the tongue lies motionless in the mouth. Articulation is impaired in proportion to the loss of movement, but very slightly in unilateral disease. Mastication also suffers at the same time. If atrophy supervenes, the tongue shrinks in bulk and feels flabby, and the mucous membrane is thrown into wrinkles. The position of the lesion is suggested by the associated symptoms. If it is above the nucleus, there may be hemiplegic weakness on the same side as the lingual paralysis, but there will not be atrophy; if below the nucleus, atrophy may ensue, and paralysis of the limbs, if any, will be on the opposite side. If the symptoms are bilateral, as in bulbar paralysis, the lesion is at or near the nuclei, and it is the same if the other lower cranial nerves are involved.

Treatment.—This must follow the casual indications. To galvanise the tongue a spatula may be used with a wooden handle, and insulated by sealing-wax where it passes over the lips (Gowers).

LESIONS OF SPINAL NERVES.

The spinal nerves arise from the spinal cord, each by two roots, an anterior motor and posterior sensory. These nerves contain the lower motor and sensory neurons, the cell-bodies of the former lying in the spinal anterior cornua, and those of the latter in the ganglia of the posterior roots. The portions of the spinal cord with which successive pairs of nerves are connected are called *segments*, and are named after the attached nerves, and not after the vertebræ opposite which each segment lies. The cervical, lumbar, and sacral nerves soon after the junction of their roots unite with one another, and lose themselves in plexuses, from which emerge the named nerve-trunks to the limbs, such as median, ulnar, sciatic, &c. From this it results that in those regions each segment of the cord corresponds to more than one distal nerve-trunk, and each distal nerve-trunk to more than one spinal segment. This fact has rendered unusually difficult the inquiry as to the relation between spinal segments (*i.e.*, nerve-roots), on the one hand, and the individual muscles and the cutaneous areas on the other. These relations have, however, been worked out by several observers (Starr, Thorburn, Head, and others), and the table and figures (pp. 214–7), which give these results, will be found of value in connection with diseases both of the nerves and of the spinal cord.

The nerves may be injured or diseased at their roots, or in the plexus or nerve-trunks beyond them. Isolated nerve paralyses in the limbs are more likely to be caused by lesions below the plexuses, while injuries of the plexuses, or nerve-roots, are more liable to be followed by grouped paralyses.

Lesions of the nerve-roots arise in connection with diseases, injuries, and tumours of the spinal cord or spinal column, and the symptoms may be combined with those of the central affection: as, for instance, in meningitis, caries, and the degenerative changes of infantile paralysis and locomotor ataxy. Lesions of the spinal nerve-trunks are mostly injuries from pressure, wounds, fractures, and dislocation in the distal parts; from new growths, aneurysms, and abscesses in the proximal portions near the spinal column. Exposure to cold may set up neuritis in isolated nerves; and the causes of multiple and peripheral neuritis, already enumerated (p. 179), must not be forgotten.

Seeing that the spinal nerves contain both motor and sensory fibres, the symptoms of their disease are both loss of muscular power and anæsthesia, determined by the distribution of the nerve-fibres to muscles and skin respectively. If the lesion is persistent, atrophy and altered electrical reactions (p. 174) of the muscles will ensue, and perhaps trophic changes in the skin.

Some of the more important and frequent lesions of these nerves may be here shortly described.

PHRENIC NERVE.

The fibres of the phrenic nerve are involved in disease of the cervical portion of the spinal cord, such as acute myelitis: occasionally the nerve is injured by wounds in the neck, and it may be pressed upon by tumours in the neck and thorax. It is not infrequently involved in the paralyses of diphtheria, alcoholism, and beri-beri, and presumably in other cases of multiple neuritis; and it may be affected in lead-poisoning. The characteristic symptom of a bilateral lesion is paralysis of the diaphragm. The breathing is effected solely by the action of the intercostal muscles, and accessory muscles of inspiration; the abdominal wall, instead of advancing during inspiration, is retracted, and it is driven out during expiration. Dyspnœa may be slight when the patient is tranquil, but movement increases the difficulty, and then the over-action of the thoracic walls becomes especially striking. In a less marked stage of paralysis the diaphragm seems to remain in a semi-inspiratory position, not contracting upon the contents of the abdomen, but it resists being drawn up into the chest, so that the abdominal wall is more stationary during respiration. In a bad case a full respiration is impossible, coughing becomes difficult, husky, or noiseless, and the voice is almost lost. Moreover, the circulation of blood is impeded in the lower part of the lungs, from inaction of the diaphragm, and here abundant mucus accumulates, which the patient is unable to expel. The danger thus arising is, of course, increased by any bronchitis. Diaphragmatic paralysis, though it sometimes gives the *coup de grâce* to alcoholic and diphtheritic paralyses, is not necessarily fatal—it may last some days or weeks, and then gradually clear up.

POSTERIOR THORACIC NERVE.

This nerve is sometimes injured, as it lies in the posterior triangle of the neck, by carrying loads on the shoulder. The lesion is thus common in porters, &c., and is nine times more frequent in men than in women; it also arises from cold, and may be seen in anterior poliomyelitis. The paralysis of the serratus magnus which results is distinguished by the position of the scapula. The inferior angle approaches the spine from the unopposed action of the rhomboidei and the levator anguli scapulæ. The arm is with difficulty raised above the horizontal, since complete elevation is largely effected in health by the serratus magnus rotating the lower angle of the scapula forwards. When the arm is moved forwards in the horizontal position, the angle of the scapula projects from the chest, so that the fingers can be placed underneath it; and it approaches the spine at the same time. Cutaneous anæsthesia is, as a rule, absent, but the onset may be accompanied by neuralgic pains. The digitations of the muscle below the axilla may be obviously wasted or inactive, as compared with the other side.

CIRCUMFLEX NERVE.

Dislocations of the shoulder, falls or blows on the shoulder, and the pressure of a crutch are the special causes of paralysis of this nerve. In lesions of the brachial plexus, in lead paralysis, and in spinal lesions it may also be involved. It is rarely caused by cold. The chief symptom is paralysis of the deltoid muscle, so that the arm cannot be raised to the horizontal position; any attempt results in elevation of the shoulder by the trapezius and serratus, while the arm hangs vertically. In old cases atrophy and reaction of degeneration supervene. Cutaneous anæsthesia is often absent, and paralysis of the teres minor, also supplied by the circumflex, cannot generally be recognised. It must be remembered that ankylosis of the shoulder-joint fixes the arm in the same position, and leads to atrophy of the muscle. Passive motion will distinguish between them.

MUSCULO-SPIRAL NERVE.

This nerve, from its exposed position as it winds round the humerus, is especially liable to injury from prolonged pressure, from the use of a crutch, or from hanging the arm over the back of a chair during sleep, or from sleeping with the whole weight of the body upon one arm. It may be also injured by fractures and dislocations, and rarely by violent action of the triceps. Cold is not a common cause. A partial affection of the branches of the musculo-spiral nerve is the characteristic feature of chronic lead-poisoning. The muscles paralysed by a lesion high up are the extensors of the elbow and wrist, the long extensors of the finger and thumb, and the supinators; but in the more common lesions a little above the elbow one or more muscles escape, especially the triceps and the supinator longus. With paralysis of the triceps there is inability to extend the forearm on the arm. This must be tested with the arm raised, so as to avoid the action of gravity in extending the forearm. The extensor paralysis of the wrist and fingers is shown by the "dropped wrist" or "dropped hand." If the forearm is extended in pronation, the hand hangs vertically, and cannot be raised, nor can the fingers or thumb be lifted from their pendant position. If the hand be raised and the first phalanges be supported, the middle and terminal phalanges can then be extended by the action of the interossei and lumbricales. The supinator paralysis prevents any movement from the position of complete pronation, but if the forearm be flexed supination will be effected by the biceps. Flexion in semi-pronation is weakened by the absence of the supinator longus, and the characteristic prominence of this muscle in movements of flexion is absent. Flexion of the fingers is considerably weakened by the passive approximation of the ends of the muscles, and a curious prominence forms on the back of the wrist, which is possibly due, as suggested by Gowers, to the synovial sacs and bones of the carpus projecting in that direction when unsupported by the extensor tendons. If there is cutaneous anæsthesia, it affects the outer side of the back of the hand, the back of the thumb, and the back of the first phalanges of the fore and middle fingers.

Erb's paralysis of the brachial plexus.—Either from injury to the neck, or from disease such as neuritis, the following muscles may be paralysed simultaneously:—deltoid, biceps, brachialis anticus, supra-spinatus, infra-spinatus, and perhaps the supinator longus. This form of paralysis is believed to be due to a lesion of the roots of the fifth and sixth cervical nerves.

ULNAR NERVE.

This is exposed to wounds and injuries in the arm, and near the wrist, and to injury by dislocations of the shoulder and elbow, and by fractures of the forearm. A neuritis from cold is not common; but the stretching of the nerve at the elbow by extreme flexion of that joint probably sets up neuritis sometimes, especially in those already out of health. The movements affected are flexion of the wrist towards the ulnar side, flexion of the fingers, especially of the first phalanges, with extension of the second and third, adduction of the thumb and the lateral movements of the fingers by the interossei. In old cases the unopposed action of the extensor muscles leads to over-extension of the first phalanges, and flexion of the second and third, producing the claw-like hand (*main en griffe*). Anæsthesia is variable: its limits are the ulnar part of the hand corresponding to one and a half fingers in front and two and a half on the back.

MEDIAN NERVE.

This is mostly affected by injuries, occasionally by neuritis. When it is paralysed the forearm cannot be pronated more than half-way, flexion of the wrist takes place towards the ulnar side, the thumb cannot be flexed or abducted, the second phalanges of the fingers cannot be flexed on the first, nor the third phalanges on the second, except in the case of the third and fourth fingers, in which this is effected by the ulnar half of the flexor profundus. Anæsthesia, if it occurs, affects the radial half of the palm of the hand, the anterior aspect of the thumb, forefinger, middle finger, and radial side of ring finger, and the dorsal surfaces of the thumb and the same three fingers (or fore and middle only in some persons) beyond the first joint.

SCIATIC NERVE.

The most common paralysis in the lower extremity is that due to disease of the sciatic nerve, wholly or in part. It may be from tumours or diseased bone in the pelvis, from dislocations of the hip, from wounds, tumours, or neuroma in the thigh. Neuritis is relatively common, and to this many, if not all, cases of *sciatica* are to be attributed. In a lesion of the sciatic trunk above the upper third of the thigh, the flexors of the leg upon the thigh are involved, in addition to those affected through the two popliteal branches.

External Popliteal (Peroneal) Nerve.

This nerve, like the ulnar, occupies an exposed position near the knee. Lesions cause paralysis of the tibialis anticus, the peronei, the long extensors of the toes, and the short extensor on the dorsum of the foot. The foot hangs down when raised from the ground (foot-drop), and lies extended when the patient is in bed; dorsal flexion of the foot, and of the toes, and abduction of the foot, and elevation of its outer border are deficient or impossible. Wasting of the anterior tibial muscles, and of the little extensor brevis can be readily recognised by contrast with the other leg; in old cases permanent extension of the foot (talipes) is produced mainly by the action of gravity, especially when the patient lies in bed. Anæsthesia affects the outer half of the front of the leg and the dorsum of the foot.

Internal Popliteal Nerve.

Extension (plantar flexion) of the foot and flexion of the toes cannot be effected. The patient cannot raise himself on his toes, nor can he adduct the foot. In old cases talipes calcaneus may develop, and a kind of claw-foot from over-extension of the proximal phalanges, and flexion of the second and third. Anæsthesia corresponds to the outer half of the back of the leg, and the sole of the foot.

Sciatica.

This has long been regarded as a typical neuralgia involving the sciatic nerve, but the fact, so commonly observed, that in old cases the muscles of the lower extremity waste, indicates that there is something more than a purely functional neuralgia; and Gowers shows that it is, indeed, in a large majority of cases, a genuine neuritis, as proved by the conditions under which it arises; by the accompanying symptoms, anæsthesia, and muscular atrophy; and by the fact that in a few cases *post-mortem* neuritis has actually been found.

Ætiology.—The disease is much more common in men than in women, and occurs in the latter half of life, especially between the ages of forty and fifty. In many cases no good cause can be shown; in others it arises in connection with gouty and rheumatic tendencies; in a large number of cases it is excited by cold; and syphilis is responsible for some others. It is also set up by mechanical causes, such as blows, and long pressure on the nerve, as by the edge of a chair; by fatigue from excessive walking, or otherwise; and by some diseases within and without the pelvis.

Fibrillation + Paraesthesia

Symptoms.—The chief symptom is pain, which is felt in the nerve-trunk or its branches; it comes on either gradually or suddenly, and is aggravated by movement or the attempt to walk, or by anything which causes the nerve to be stretched or pressed upon. To avoid this, the patient holds the leg fixed at the knee when walking, and when lying down the most comfortable position is one of flexion. The pain is most often in the back of the thigh, but may extend down the back of the calf, along the outer side of the leg, and to the sole of the foot; and is often most intense at certain spots—namely, near the posterior iliac spine, at the sciatic notch, about the middle of the thigh, behind the knee, below the head of the fibula, behind the external malleolus, and on the dorsum of the foot. The nerve, too, is tender to pressure, especially at the sciatic notch, along the back of the thigh, and in the external popliteal branch behind the head of the fibula.

The pain is burning or gnawing, more or less continuous, but intensified by movement or manipulation. In severe cases, other disturbances of nerve-function occur. These are tingling, formication, and anæsthesia, in connection with sensory fibres; and atrophy of muscles, muscular weakness, and sometimes fibrillary tremors, from implication of motor fibres. The electrical reactions are not markedly altered except in severe cases, when reaction of degeneration may occur.

Paraes
Fibrillat

The duration is very variable. Slight cases may recover quickly; severer cases last months or years; and after subsidence of the pain, the muscular wasting, fibrillary contractions, and a tendency to cramps may persist for some time.

Diagnosis.—The diagnostic points in favour of sciatic neuritis are the tenderness of the nerve and the presence of anæsthesia and muscular atrophy. In these it differs from a pure neuralgia of this region. Further, it must be distinguished from the pains due to hip-joint disease, sacro-iliac disease, pelvic lesions, and tumours of the femur, in which, also, tenderness of the nerve should be absent, and the pains more limited to the seat of the lesion. But even if neuritis is present, it may be secondary to such lesions, and the symptoms special to them should be carefully looked for before concluding that the disease is a primary sciatica.

Prognosis.—This is, on the whole, favourable.

Treatment.—Complete rest is essential, in the position that most eases the pain. Any gouty disposition may be met by suitable remedies, such as saline diuretics and purges. In acute cases—especially those due to cold—hot poultices or fomentations should be applied to the affected limb, and internally, perchloride of mercury or iodide of potassium should be given. In later stages, both counter-irritants and sedatives are of value. Mustard plasters, blisters frequently repeated, acetic turpentine liniments, and the chloride of methyl spray as recommended by Debove may all give some relief. This last is only a method of obtaining counter-irritation by temporary freezing of the skin along the course of the nerve. Often the greatest benefit is obtained from morphia injections, but they must be given with proper fear that a morphia habit may be subsequently induced. The injection of ½ or 1 grain of cocaine is also said to be efficacious. Belladonna, opium, and chloroform liniments may be used, and galvanism in the same way as is described under neuralgia. Acupuncture along the course of the nerve is often of value. Finally, in severe cases, nerve-stretching remains as a means of getting relief, generally for some time, even if the pain subsequently recurs.

SPASMS OF MUSCLES SUPPLIED BY CRANIAL NERVES.

Spasm of Ocular Muscles.

This occurs in association with various diseases of the eye, in the conjugate deviation of cerebral disease, in hysteria, and other conditions. Clonic spasm is termed *nystagmus*. The eyes are moved rapidly to and fro, generally in a lateral direction, sometimes vertically. It results from some ocular defects, such as extreme choroidal atrophy, and albinism. It occurs in a number of central nervous diseases, with greatest constancy in disseminated sclerosis and Friedreich's ataxy, but also frequently in tumours of the cerebellum. It also appears in miners, especially in those who work in the recumbent position.

Spasm of the Jaw.

Trismus, or spasm of the muscles closing the jaw, is one of the first indications of the onset of tetanus. The jaw is fixed by tonic contraction of the masseter or temporal muscles, so that the teeth cannot be separated more than a few lines. A similar spasm may be due to irritation of the teeth, or to stomatitis, or, on the other hand, to central disease, such as disease of the pons in the neighbourhood of the fifth nerve-nucleus. It must be distinguished from tumours or rheumatic arthritis fixing the jaw-joint. *Clonic* spasm of the jaw occurs in rigor, in convulsions, and in hysteria—rarely as an isolated phenomenon.

Facial Spasm.

Irregular contractions of the facial muscles takes place in chorea, and a tonic contraction is a late stage of facial paralysis. Boys and girls often acquire a habit of twitching certain muscles of the face, neck, or other part of the body, and this habit may last into adult life. More serious cases of facial spasm (*convulsive tic, histrionic spasm*) occur in people over twenty years of age, and mostly between thirty and sixty. In some of these there is actual irritation of the facial nerve by tumours in the pons, or of the facial cortical centre on the opposite side of the brain; in most cases the condition seems to be functional. It is much more frequent in women than in men, and arises from emotion, mental anxiety, irritation of the peripheral branches of the fifth nerve as in the eyelids, or the teeth, and from exposure to cold. The spasm chiefly affects the orbicularis palpebrarum and the zygomatici, so that the eye is half closed, and the angle of the mouth is drawn up. Other facial muscles, including the platysma myoides, are also contracted, but the orbicularis oris and frontalis muscle, as a rule, escape. The contractions are momentary, and frequently repeated; or the spasm is of longer duration, and recurs at longer intervals; but it causes no pain. The spasm is at first entirely on one side, and only in severe or prolonged cases affects the other side. The electrical reactions are usually normal.

The **Prognosis** in a well-established case is unsatisfactory; the disease will last months, or years, and even to the end of life.

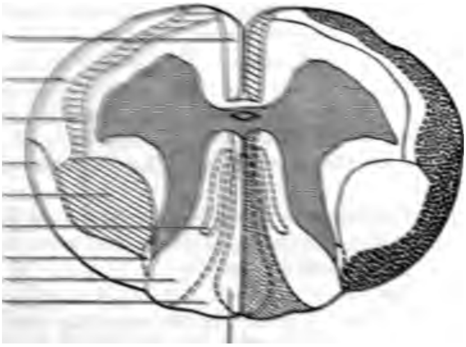
The **Treatment** consists in the removal of causes of irritation, if they can be recognised; the use of nervine tonics, such as zinc sulphate, iron perchloride or sulphate, and strychnine; and of sedatives, especially the hypodermic injection of morphia. A weak galvanic current applied continuously, and counter-irritation by blisters behind the ear, may be tried. Nerve-stretching has been performed in some cases, but only exceptionally with any lasting benefit.

DISEASES OF THE SPINAL CORD.

PRELIMINARY CONSIDERATIONS.

It will not be necessary to go into full detail as to the anatomy of the spinal cord. The familiar appearance of gray cornua and white columns as seen on transverse section is shown in the accompanying figure, and the further subdivisions of the white columns are there indicated.

The *pyramidal tract* descends through the brain from the cortex, enters the spinal cord, partly decussating in the medulla oblongata, [illegible] the middle line. The un-[illegible] column of [illegible] size from above downwards. [illegible] region. The other portion, the [illegible] posterior half of the lateral [illegible] posterior cornu. It reaches [illegible] region, but higher up it lies [illegible] tract consists of the [illegible] neurons, the terminal [illegible] motor nerve-cells) [illegible]



[illegible] Spinal Cord, showing Tracts of White Matter. (After Sherrington.)

[illegible] *postero-external*, or column of Burdach, [illegible] column of Goll) contain chiefly the axis-[illegible] sensory neurons, on the same side [illegible] The *[illegible]-lateral ascending* tract begins [illegible] contains axis-cylinder processes of [illegible] neurons the cell-bodies of which lie in [illegible] the opposite side of the cord: they [illegible] The *cerebellar tract* begins in the [illegible] contains the axis-cylinder pro-[illegible] the cell-bodies form Clarke's [illegible] the cerebellum. It is known [illegible] downwards by the *antero-[illegible] tract.*

[illegible] disease the cord is, first, a [illegible] between the brain and the limbs [illegible] is effected by the system of [illegible] neurons already described (*see* also p. 164). Secondly, the cord is a centre for reflex action, by means of the cell-bodies of the lower motor neurons contained in the anterior cornua. Thirdly, these nerve-cells at the same time control the nutrition of the muscles, while the nutrition of the skin and other parts seems to be related to the sensory neurons.

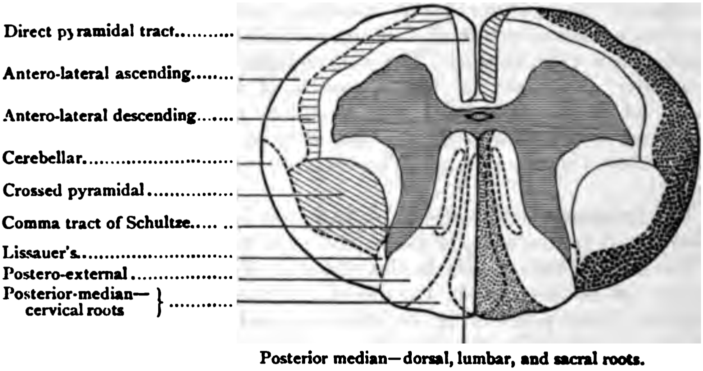
If the *gray cornu*, at any level, be alone affected by any lesion, the immediate result is paralysis of the muscles in connection with it, and loss of reflexes in the corresponding area. If the lesion is severe or protracted, degenerative effects take place in the nerve, of the same kind as those which follow neuritis and lesions of the nerves, and as a consequence atrophy of nerves, atrophy of muscles, altered electrical reactions and nutritive changes are observed.

If a lesion is confined to the *white column*, there will be loss of conduction of motor or sensory impulses according to the situation of the lesion—that is to say, there will be paralysis or anæsthesia, or other form of sensory disturbance, or inco-ordination of movement, or some combination of these. But so long as the gray centres (and anterior root fibres) are untouched there will be no pronounced atrophy and no degenerative reactions.

But the white columns of the cord consist of nerve-fibres, and just as the nerve-trunks themselves degenerate when their fibres are cut off from their centres, so in the cord a *secondary degeneration* takes place, when certain tracts are affected, so as to interrupt their connection with their functional centres. This degeneration takes place in the direction of conduction of impulses—that is, downwards (or peripherally) in the case of motor neurons (descending degeneration); and upwards (or centripetally) in the case of sensory neurons (ascending degeneration). Thus, a lesion in the centre of the cord destroying or severely compressing the white columns causes degeneration of the pyramidal tract, direct and crossed, in the part of the cord below the lesion, and sometimes of the small comma tract of Schultze; and of the posterior median column, cerebellar tract, and antero-lateral ascending tract in the part of the cord above. The postero-external column also degenerates for a short distance above the lesion; but the cerebellar tract is unaffected by lesions below the junction of the lumbar and dorsal portions. As will be shown later, unilateral cerebral lesions involving the pyramidal tracts also cause a descending degeneration of the pyramidal tract, which affects the direct tract on the same side, and the crossed tract on the opposite side of the cord; obviously in such a case the sensory columns are not affected. To the naked eye the areas of secondary degeneration are of a reddish or yellowish-gray colour, but in early stages they may be scarcely visible. They may then be demonstrated by various staining reagents.

partly continuing on the same side of the middle line. The undecussated portion is the *direct* pyramidal tract, or column of Türck. It gradually diminishes in size from above downwards, and is not present in the lumbar region. The other portion, the *crossed pyramidal tract*, occupies the posterior half of the lateral column, outside and in front of the posterior cornu. It reaches the surface of the cord in the lumbar region, but higher up it lies within the cerebellar tract. The pyramidal tract consists of the axis-cylinder processes of the upper *motor* neurons, the terminal arborisations of which lie around the cell-bodies (motor nerve-cells) of the lower motor neurons in the anterior cornua.

FIG. 11.



Transverse Section of Spinal Cord, showing Tracts of White Matter. (After *Sherrington.*)

The posterior columns (*postero-external*, or column of Burdach, and *posterior median*, or column of Goll) contain chiefly the axis-cylinder processes of the lower sensory neurons, on the same side as their cell-bodies. The *antero-lateral ascending* tract begins above the lumbar cord; it contains axis-cylinder processes of middle (or upper) sensory neurons, the cell-bodies of which lie in the posterior gray cornua on the opposite side of the cord: they are therefore decussated tracts. The *cerebellar tract* begins in the middle of the lumbar region: it contains the axis-cylinder processes of sensory neurons, of which the cell-bodies form Clarke's columns; and the fibres terminate in the cerebellum. It is known further that impulses are transmitted downwards by the *antero-lateral descending tract* and by the *comma tract.*

Effects of Lesions.—In relation to disease the cord is, first, a means of transmitting impulses between the brain and the limbs and other parts of the body, and this is effected by the system of neurons already described (*see* also p. 164). Secondly, the cord is a centre for reflex action, by means of the cell-bodies of the lower motor neurons contained in the anterior cornua. Thirdly, these nerve-cells at the same time control the nutrition of the muscles, while the nutrition of the skin and other parts seems to be related to the sensory neurons.

If the *gray cornu*, at any level, be alone affected by any lesion, the immediate result is paralysis of the muscles in connection with it, and loss of reflexes in the corresponding area. If the lesion is severe or protracted, degenerative effects take place in the nerve, of the same kind as those which follow neuritis and lesions of the nerves, and as a consequence atrophy of nerves, atrophy of muscles, altered electrical reactions and nutritive changes are observed.

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But the white columns of the cord consist of nerve-fibres, and just as the nerve-trunks themselves degenerate when their fibres are cut off from their centres, so in the cord a *secondary degeneration* takes place, when certain tracts are affected, so as to interrupt their connection with their functional centres. This degeneration takes place in the direction of conduction of impulses—that is, downwards (or peripherally) in the case of motor neurons (descending degeneration); and upwards (or centripetally) in the case of sensory neurons (ascending degeneration). Thus, a lesion in the centre of the cord destroying or severely compressing the white columns causes degeneration of the pyramidal tract, direct and crossed, in the part of the cord below the lesion, and sometimes of the small comma tract of Schultze; and of the posterior median column, cerebellar tract, and antero-lateral ascending tract in the part of the cord above. The postero-external column also degenerates for a short distance above the lesion; but the cerebellar tract is unaffected by lesions below the junction of the lumbar and dorsal portions. As will be shown later, unilateral cerebral lesions involving the pyramidal tracts also cause a descending degeneration of the pyramidal tract, which affects the direct tract on the same side, and the crossed tract on the opposite side of the cord; obviously in such a case the sensory columns are not affected. To the naked eye the areas of secondary degeneration are of a reddish or yellowish-gray colour, but in early stages they may be scarcely visible. They may then be demonstrated by various staining reagents.

The process of degeneration is similar to what takes place in the nerves: destruction of myelin, disappearance of axis-cylinders, increase of connective tissue (or neuroglia), and later the formation of granule-corpuscles—large cells filled entirely with small granules of fat, as a result of which they have a dark colour on ordinary examination, but disappear in the usual processes for clearing sections. In long-standing cases the connective tissue develops, the parts become hard and dense, and the name *sclerosis*, which is scarcely suitable in earlier stages, may be properly used.

Certain functional changes result from lesions of the pyramidal tract. These are: (1) Rigidity of the muscles corresponding to the part of the spinal cord below the lesion; (2) increased reflex irritability. They are commonly attributed to the secondary degeneration, and do, indeed, show themselves, not immediately, but after an interval, which corresponds closely with the time required for the occurrence of the change in the nerve. But the sclerosis acts probably, not by irritating the anterior cornua, but by the removal of the inhibitory control of the higher centres, so that the lower centres are unduly excitable.

The increased reflexes take place in the part of the spinal cord which is itself healthy. The knee-jerk is excessive; foot-clonus is easily produced; and sometimes the knee-jerk is followed by a temporary clonus. Pinching the skin causes retraction of the limb after a definite interval.

No symptoms have hitherto been referred to the *ascending degeneration* which follows a lesion affecting the sensory tracts.

From the above it will be seen that, with respect to motor disturbances, the results depend upon the relation of the lesion to the upper and lower neurons. If the lower neurons contained in the anterior cornua and anterior roots are injured, the condition is comparable to that following lesions of a motor nerve (*see* Neuritis). Voluntary motion, spinal reflexes, nutrition and electrical reactions are all dependent upon the integrity of the gray cornua, or the motor fibres. Hence lesions of these parts destroy these functions, and, as a result, there occur paralysis, loss of reflexes, degeneration of nerves, atrophy of muscles, and reaction of degeneration. If the upper neurons contained in the pyramidal tracts are alone injured, voluntary motion is indeed lost, because the conducting fibres from the brain to the spinal centres are interrupted; but the functions which depend solely on the integrity of the gray matter, nerve-roots, and fibres are maintained—namely, the nutrition of nerves and muscles and their electrical reactions. The spinal reflexes are, in most cases, increased.

TRANSVERSE LESIONS.

The spinal cord, having an elongated form, is naturally liable to lesions affecting its whole thickness, such as may occur from external pressure, the indiscriminate growth of tumour, or the diffuse spread of inflammation. In such cases, gray and white matters are equally affected. The results of such a transverse lesion show themselves mainly as an interruption of the *conducting* power of the cord; but if the lesion is at all extensive vertically, its effects upon the *nerve-centres* must also be considered—that is, both upper and lower neurons are affected. The results also vary, according as the lesion is ***bilateral*** or ***unilateral***, in consequence of differences between the motor and sensory fibres in the process of decussation.

RESULTS OF A BILATERAL TRANSVERSE LESION AS AFFECTING CONDUCTION.

Paralysis of all muscles below the lesion.

Anæsthesia of parts below.

Functions of bladder and rectum impaired. Trophic relations and electrical reactions continue normal.

Subsequently, muscular rigidity and increased reflexes.

This last statement requires modification, for when a transverse lesion of the spinal cord is *complete*, so as to interfere absolutely with all impulses passing through the lesion, the deep reflexes are not increased, but are abolished, and the muscles are flaccid (Bastian and others). Nevertheless, in most cases of transverse myelitis, perhaps because the lesion is incomplete, the reflexes are increased and the muscles rigid.

RESULTS OF A UNILATERAL TRANSVERSE LESION AS AFFECTING CONDUCTION.

On the same side as the lesion.	On the opposite side.
Paralysis of muscles below.	Anæsthesia of skin below.
Hyperæsthesia of skin below.	
Diminished muscular sense and sensibility.	Normal muscular power, muscular sense, reflex action, and temperature.
Reflexes at first lessened, then increased.	
Vasomotor paralysis and elevation of temperature.	
Nutrition and electrical reactions retained.	

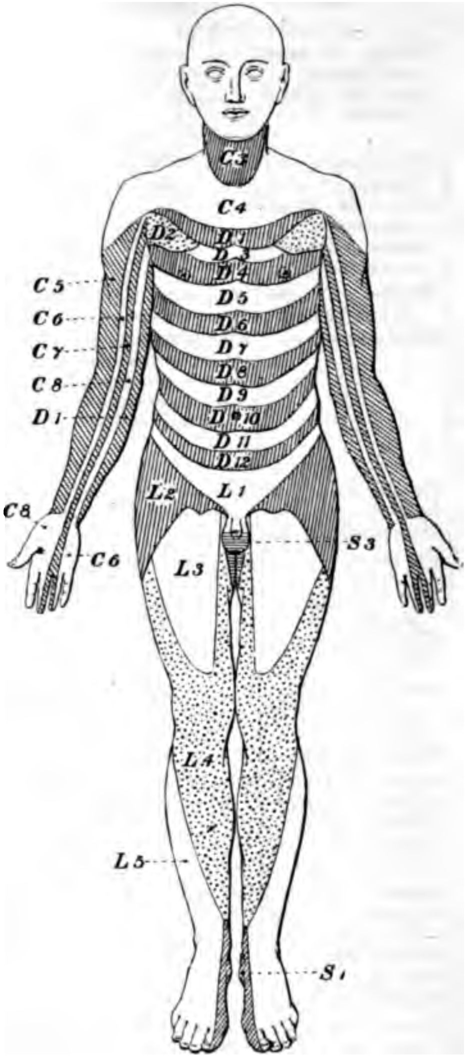
To these symptoms in each case may be added those which are due to the changes at the level of the disease.

In a total transverse lesion, these are:—paralysis, atrophy,

SEGMENT.	MUSCLES.	REFLEX AND CENTRES.	SENSATION.
First Cervical.	Rectus lateralis. Rectus capitis anticus and posticus. Sterno-hyoid. Sterno-thyroid.		
Second and Third Cervical.	Sterno-mastoid. Trapezius. Scaleni and neck. Omo-hyoid. Diaphragm.	Hypochondrium (?) Sudden inspiration produced by sudden pressure beneath the lower border of the ribs.	Back of head to vertex. Neck.
Fourth Cervical.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infraspinatus.	Pupillary (fourth cervical to second dorsal). Dilatation of the pupil produced by irritation of the neck.	Neck. Shoulder, upper surface. Outer surface of arm. Anterior thorax as far as second rib.
Fifth Cervical.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular (fifth cervical to first dorsal). Tendon reflexes of corresponding muscles.	Back of shoulder and arm. Outer side of arm and forearm to the wrist.
Sixth Cervical.	Biceps. Brachialis anticus. Extensors of hand and fingers. Pectoralis (clavicular part). Serratus magnus. Triceps. Pronators.	Tendon reflexes of extensors of the arm and forearm. Posterior wrist (sixth to eighth cervical). Tapping tendon causes extension of hand.	Back and front of arm and forearm within above. Radial side of hand to middle of second finger.
Seventh Cervical.	Triceps (long head). Extensors of hands and fingers. Pronators of hand. Flexors of hand. Subscapular. Pectoralis (costal part). Latissimus dorsi. Teres major.	Palmar (seventh cervical to first dorsal). Stroking palm causes closure of fingers.	Back and front of arm and forearm within above, and hand to middle of third finger.

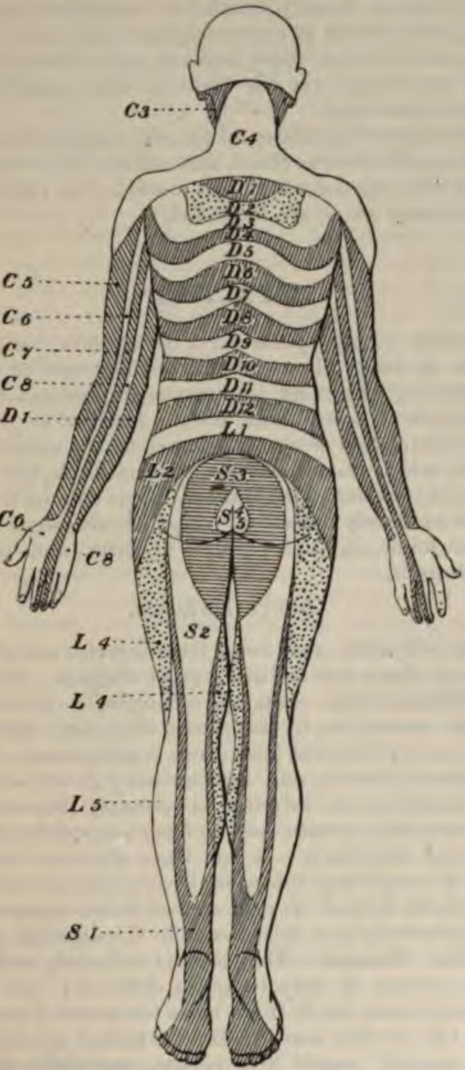
SEGMENT.	MUSCLES.	REFLEX AND CENTRES.	SENSATION.
Eighth Cervical.	Flexors of hand and fingers. Intrinsic hand muscles.	—	Back and front of arm and forearm within above. Ulnar area of hands, back and palm.
First Dorsal.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar muscles.	—	Inner side of arm and forearm to wrist.
Second to Twelfth Dorsal.	Muscles of back and abdomen. Erectores spinæ.	Epigastric (fourth to seventh dorsal). Abdominal (seventh to eleventh dorsal). Vasomotor centres (second dorsal to second lumbar).	Skin of chest, back, and abdomen, in bands running around and downwards, corresponding to spinal nerves. Upper gluteal region.
First Lumbar.	Ilio-psoas. Sartorius. Abdominal muscles.	Cremasteric (first to third lumbar).	Skin over groin and front of scrotum.
Second Lumbar.	Ilio-psoas. Sartorius. Flexors of the leg (Remak). Quadriceps femoris.	Patellar tendon (second to fourth lumbar).	Outer side and upper front of thigh.
Third Lumbar.	Adductors and inward rotators of thigh. Flexors of thigh.	—	Front and inner side of thigh.
Fourth Lumbar.	Adductors of thigh. Abductors of thigh. Tibialis anticus. Flexors of leg (Ferrier).	Gluteal (fourth to fifth lumbar).	Inner side of thigh and leg to ankle.
Fifth Lumbar.	Outward rotators. Flexors of leg (Ferrier). Flexors of foot. Peronei. Extensors of toes.	—	Outer side of leg, dorsum of foot, and outer part of sole.
First and Second Sacral.	Flexors of foot and toes. Peronei. Small muscles of foot.	Plantar (fifth lumbar to second sacral).	Back of buttock, thigh, and leg, inner side of foot and sole.
Third to Fifth Sacral.	Muscles of the perineum.	Tendo Achillis. Vesical centre. Anal centre.	Sacral region, anus, perineum, and genitals.

FIG. 12.



The figure represents some of the opinions which have been expressed as to the areas of skin corresponding to the sensory roots of the Spinal Nerves. It may be compared with the fourth column on pp. 214–215 (*see Brain*, 1893, 1894; *Amer. Journ. of Med. Sci.* 1888, 1892).

FIG. 13.



The figure represents some of the opinions which have been expressed as to the areas of skin corresponding to the sensory roots of the Spinal Nerves. It may be compared with the fourth column on pp. 214–215 (*see Brain*, 1893, 1894; *Amer. Journ. of Med. Sci.* 1888, 1892).

reaction of degeneration, and loss of reflexes in the muscles supplied by nerves directly connected with the injured segment; anæsthesia, with a band of hyperæsthesia at the same level.

In a one-sided lesion, the symptoms just enumerated occur on the side of the lesion; on the opposite side, anæsthesia alone is found at the same level.

In the accompanying table and figures, compiled from the work of Allen Starr, Thorburn, Head, and others, the relations of the segments of the spinal cord to the muscles, the reflexes, and the cutaneous sensory areas, are indicated.

MYELITIS.

Inflammation of the spinal cord, or myelitis, occurs in several forms. As an acute disease, it may be a *general* or *disseminated* myelitis, affecting white and gray matter indiscriminately throughout the cord; it is much more often a *localised* or *focal* myelitis. It is, in a certain class of cases, confined to the anterior cornua of gray matter, and is then known as *polio-myelitis*, the pathological cause of acute or atrophic spinal paralysis (infantile and adult): this will be separately described. Myelitis also occurs in a chronic form, often following upon acute myelitis, and not differing materially from it.

Acute Myelitis.

Ætiology.—Myelitis may arise from injuries and strains of the back, such as those due to lifting great weights. It is set up by adjacent inflammations, such as meningitis; it is sometimes caused, with meningitis, by bed-sores ulcerating into the spinal canal; and a very frequent local cause is compression of the spinal cord by tumours, caries, and inflammatory growth in the membranes. Its relation to infection is becoming increasingly important: it occurs after certain specific fevers, especially enteric fever, small-pox, and diphtheria; it has been observed within a few months, or a year, after infection by syphilis, and in some other cases, apparently related to cold or wet, micro-organisms (streptococci and diplococci) have been found in the diseased parts.

Anatomical Changes.—The cord is softened, and bulges on transverse section, or may be quite diffluent; but previous to section it may seem hard, from mere tenseness of the membrane containing the swollen cord.* To the naked eye the section is congested, minute vessels are visible, especially in the gray matter, and the gray matter itself is darker than usual, and its outline is indistinct. Patches of gray tint may be present in the white column, or the whole surface is confused. Under the microscope, in early stages, are found perhaps capillary hæmorrhages; and leucocytes in the lymphatic sheaths and around the vessels. The nerve-fibres suffer by breaking up of their myelin, and here and there occurs a peculiar change—hypertrophy of the axis-cylinders. This consists in a fusiform enlargement, which in its thickest part is five or six times the diameter of the normal axis-cylinder. The nerve-cells are swollen, granular, with perhaps fatty globules, and some of them undergo vacuolation. Subsequently both nerve-cells and fibres degenerate, the connective tissue increases in quantity, the cells, known as Deiter's cells, are larger and more numerous, and granule-cells are formed in abundance. Finally, in cases of long-standing, or going on into a chronic condition, the granule-cells disappear also, and the new connective tissue becomes firm, dense, even finely fibrous, so that a definite *sclerosis* is the result. It is possible that some cases of so-called myelitis are thrombotic or embolic in origin, and not true inflammations. An actual *abscess* of the spinal cord is very rare.

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* It must not be forgotten that any cord may become soft from decomposition, or may be reduced to a pulp by clumsiness in extracting it from the spinal column.

The extent of the lesion is variable; and the following forms are distinguished:—*Focal or localised myelitis*, when the lesion is very limited in extent; *diffuse myelitis*, when it is of considerable extent continuously; *disseminated myelitis*, when there are numerous scattered patches over a large extent; *transverse myelitis*, when the lesion extends across the cord and is of little vertical extent. *Central* and *peripheral myelitis* are also spoken of.

A localised or transverse myelitis persisting for a certain time is followed by ascending and descending secondary degenerations.

Symptoms.—Myelitis mostly begins with numbness or tingling in the extremities, and in acute cases there may be malaise or slight febrile reaction; or there are painful sensations in the extremities, or hyperæsthesia, or pains in the back or muscles. Twitchings and tremors of the muscles, or cramps and spasms, may also occur as early symptoms, indicating a stage of irritation. Within a day or two, or even in a few hours in the most acute cases, paralysis and anæsthesia become well marked. The symptoms vary somewhat according to the extent of cord involved.

Acute diffuse, and disseminated myelitis are comparatively rare. Numbness and tingling, weakness, and then paralysis rapidly develop. The patient is more less completely paralysed in all four limbs, and in the abdominal and intercostal muscles; there are general anæsthesia, loss of reflexes, retention of urine, and involuntary passage of fæces. The temperature rises to 102° or 103°; the breathing becomes seriously involved, and death often takes place in from three days to three or four weeks. It is the disseminated form which is more likely to occur after specific fevers. It is occasionally accompanied by optic neuritis.

A form of disseminated myelitis, combined with a similar lesion of the brain, is described under the name of *acute disseminated encephalo-myelitis* (*acute central ataxia* of Leyden); numerous small foci of inflammation are found in the cord, pons, and the cerebrum.

The disease nearly always occurs during or soon after an infectious disease or similar process; consequently a large proportion of the cases are in children. The onset is sudden and suggests the implication of the whole cerebral nervous system. There is often unconsciousness, either with paralysis, or with restlessness and delusion; and after a time the unconsciousness clears up, and a stage is reached which is characterised by ataxia or choreic movements. In other cases the sudden cerebral symptoms are absent, and ataxia is the first symptom. In this stage there are ataxic movements or intention-tremors of the legs, arms, and head; disorders of speech, especially of the scanning variety, but sometimes dysarthric; and sometimes disturbances of intellect. The gait is variable; it may be ataxic, or spastic, or paretic, or tremulous. The arms are less commonly ataxic, but more often present intention-tremor; and the same occurs with the head. There is great variety in the subsequent course: a few cases recover quickly and permanently; many recover after months, or after one or two relapses; in other cases, some symptoms, especially speech disturbances, persist; but death is rare. Aphasia or optic neuritis occasionally occurs in the earliest stage. The resemblance in many points to disseminated sclerosis (*see* p. 251) is obvious, but nystagmus appears to be uncommon.

In *localised* and *transverse myelitis* the symptoms will depend somewhat on the part of the cord affected. It is often situate in the *mid-dorsal* or *lower dorsal* region. The characteristic symptoms then are paralysis and anæsthesia of the lower extremities, constituting the ordinary form of *paraplegia*. In severe cases, the patient lies in bed, unable to raise the leg from the bed, or even move a toe. There is more or less complete numbness and loss of sensation in the legs, thighs, and lower part of the abdomen, up to a transverse line about the level of the umbilicus, higher or lower according to the exact position of the lesion. This upper limit of anæsthesia is generally sharply defined, and at the same level there is often a narrow band of *hyperæsthesia*, and a painful sensation extends from the seat of the lesion round each side of the body to the front. This is known as *girdle-pain*. The reflexes are commonly increased. Constipation is usually present; the bladder may be at first paralysed, and retention occurs. After a short time, since the lesion is above the spinal centres for the bladder, the power of expulsion is restored, but from interruption of the communication between the bladder and the brain the sensation of distension is not experienced, and outline is indistinct. Patches of gray tint may be present in the white column, or the whole surface is confused. Under the microscope, in early stages, are found perhaps capillary hæmorrhages; and leucocytes in the lymphatic sheaths and around the vessels. The nerve-fibres suffer by breaking up of their myelin, and here and there occurs a peculiar change—hypertrophy of the axis-cylinders. This consists in a fusiform enlargement, which in its thickest part is five or six times the diameter of the normal axis-cylinder. The nerve-cells are swollen, granular, with perhaps fatty globules, and some of them undergo vacuolation. Subsequently both nerve-cells and fibres degenerate, the connective tissue increases in quantity, the cells, known as Deiter's cells, are larger and more numerous, and granule-cells are formed in abundance. Finally, in cases of long-standing, or going on into a chronic condition, the granule-cells disappear also, and the new connective tissue becomes firm, dense, even finely fibrous, so that a definite *sclerosis* is the result. It is possible that some cases of so-called myelitis are thrombotic or embolic in origin, and not true inflammations. An actual *abscess* of the spinal cord is very rare.

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(tetanus, meningitis, fractured spine), *hyperpyrexia* occurs, the temperature rising to 107°, 108°, or 110° F.; and *priapism* is occasionally observed.

Other symptoms of cervical lesion are contraction of the pupil, and diminution of the palpebral fissure, dysphagia, hiccough, and very slow or very quick pulse.

Variations of the symptoms may also occur as a result of the myelitis being limited to one side, or to one small portion of the transverse section; or two or more patches of myelitis may occur in different parts of the cord.

Death in cases of acute myelitis takes place (1) from pulmonary complications following upon paralysis of the respiratory muscles; (2) from bed-sores intensifying exhaustion, or leading to pyæmia; (3) from vesical complications; (4) from exhaustion; (5) from intercurrent affections, such as pneumonia or bronchitis. The bladder is extremely liable to cystitis, partly from trophic disturbance, partly from retention of urine. Sometimes, the use of a catheter is responsible for the introduction of organisms which may set up septic inflammation. When cystitis occurs the urine rapidly decomposes and becomes ammoniacal, unless it is repeatedly removed by the catheter; it contains pus, or muco-pus, and readily deposits crystals of ammonio-magnesian phosphate on standing. Almost at any time the septic condition of the bladder may extend up the ureters to the kidneys, and suppurative pyelitis and nephritis will then occur, and the patient may die with uræmic symptoms. Sometimes death occurs from the condition which has been described as "catheter fever" and "urethral fever."

Cases which escape these dangers generally lapse into a chronic condition, which may be of indefinite duration, but sometimes recovery slowly takes place after many months. And a small number of cases get well comparatively quickly. Myelitis from compression in caries of the spine often gets better; acute diffuse myelitis is mostly fatal.

Diagnosis.—Myelitis may be confounded with spinal meningitis, hæmorrhage into the spinal cord or membranes, multiple neuritis, and hysterical conditions.

In *spinal meningitis* there is usually more evidence of irritation of the nerve-roots, shown by radiating and local pains, hyperæsthesia, and muscular spasm; and febrile reaction is more constant and persistent. *Hæmorrhage* into the spinal cord is generally quite sudden, severe local pain being quickly followed by paralysis below the lesion, whereas in myelitis, as a rule, the paralysis develops more gradually, with some preceding symptoms of sensory or motor irritation; febrile reaction may be also present for a few days in myelitis. In *meningeal hæmorrhage* the onset is again sudden, and the signs of irritation of the nerve-roots are more pronounced than in myelitis. Cases of *multiple neuritis* have no doubt been frequently regarded as instances of myelitis. The points in favour of neuritis are the affection of all four limbs simultaneously, the early predominance of extensor paralysis of the hands and feet, and the implication of the face and larynx in some cases; the co-existence, in the parts affected, of anæsthesia with muscular atrophy, altered electrical reactions, and diminished reflexes, showing a universal interference with sensory and motor paths at or below the trophic centres, such as can only occur from lesions of many nerve-trunks, or a diffuse lesion of the cord throughout its whole length. In neuritis there are also tenderness of nerve-trunks in exposed situations, and tenderness of the muscles, especially those of the calf. A history of alcoholism, or the mental condition previously described (*see* p. 181), is in favour of neuritis, and a very gradual onset is perhaps more frequent in neuritis than in myelitis.

From *hysterical paralysis* the diagnosis is often most difficult; the history of the patient, previous to hysterical attacks, or the first appearance of the symptoms after some emotional disturbance may justify a suspicion of hysteria, but the case must be carefully examined from the point of view of structural lesions, since hysterical people are not excluded from the possibility of organic disease. Patients with hysterical paraplegia do not develop the rigidity and increased reflexes of transverse myelitis, nor do they get atrophy of the muscles, bed-sores, incontinence of urine and fæces, or cystitis; nor do they have girdle-pain. It is especially slight cases that are apt to be set down as hysteria; where the patient can perhaps stand, but her inability to put one leg before the other is attributed to unwillingness. A careful examination of the muscular power while sitting or in bed, frequently repeated to test its constancy, and the use of electrical tests, will probably guard against this. It must be allowed that a good deal of weakness of the lower extremities may arise in persons who are not hysterical, from purely *functional* causes, such as exhaustion from long illnesses, or prolonged over-exertion, when it may be supposed that the functions of the cord itself are at fault rather than those of the brain as in pure hysteria.

When other diseases have been excluded, it yet remains to form an opinion as to the position and origin of the myelitis. Its localisation may be determined by comparing the symptoms with the table and figures (*see* pp. 214–217). A few scattered foci of myelitis may be due to syphilis. But the most important thing is to examine for caries of the spine, by asking for a history of blow or strain, and looking for any undue prominence of a vertebral spine; and this frequently, for paraplegic symptoms may precede by several months the first appearance of angular curvature.

Treatment.—Rest is, of course, essential, and in severe cases, or cases likely to be of long duration, a water bed should be provided to avoid the risk of bed-sores. Different kinds of local treatment have been recommended: the application of hot fomentations, hot-water bags, mustard plasters, or stimulating liniments; the abstraction of blood by leeches, or the use of dry-cupping. On the other hand, ice-bags are sometimes applied to the spine. They are only likely to do good in the early stages. In cases due to exposure to cold, Gowers recommends a hot bath, followed by free diaphoresis; and he suggests the prone position, where practicable, in order to avoid the congestion which is due to gravitation.

Internally, potassium iodide and mercuric chloride are frequently given, but without much evidence that they have any effect in lessening the inflammation. Even in cases due to syphilis, their influence does not seem to be so marked as in ordinary tertiary lesions; but mercury should be well tried when the myelitis occurs within the period of the "secondary" stage. Ergot, or ergotin, injected subcutaneously, is supposed to diminish vascular engorgement, and may perhaps be most useful if hæmorrhage has any share in the lesion. Sodium salicylate may very properly be tried in cases apparently owning a toxic or febrile origin. The greatest care must be taken to prevent bed-sores by relieving the pressure on prominent parts, by keeping the skin perfectly clean, washing it daily with spirit lotion, dusting the sheet beneath it with oxide of zinc or starch powder, and changing this whenever it becomes moist from any cause. The bladder, also, must be constantly attended to. If the urine is retained, it must be drawn off with the catheter two or three times daily, the instrument being carefully cleaned on each occasion. If cystitis occurs, and the urine becomes alkaline and offensive, antiseptic injections may be used; and salicylic acid has been given internally with the same object.

The diet should be light and nutritious, and the bowels should be kept active. If, from advancing disease, mucus accumulates in the bronchial tubes, carbonate of ammonium will sometimes clear the chest in a remarkable manner; but may, of course, have only a temporary effect.

In the later stages, tonics—such as quinine, arsenic, iron, strychnine—should be given. If the limbs are flaccid, galvanism or faradism may be of service; but it is of less value, or not advisable in cases with rigidity, well-nourished muscles, and increased reflexes, when its use may unduly excite the reflex action of the muscles.

Chronic Myelitis.

This term might be supposed to include (1) cases of myelitis that arise slowly and gradually, (2) unrecovered cases that have begun as acute myelitis, and (3) the different varieties of *sclerosis*, which are, no doubt, primarily degenerative. But it is believed by some authors that all cases which have been regarded as belonging to the first class are really instances of the multiple or disseminated sclerosis, which will be described shortly (*see* p. 251).

Chronic myelitis, like the acute form, may be transverse or focal, disseminated or diffuse. Cases believed to be primarily chronic have appeared to arise sometimes from syphilis, repeated exposure to cold, alcohol, or chronic lead poisoning. It may follow upon spinal meningitis, and then affects the surface of the cord adjacent to the membranes, forming a *peripheral* myelitis.

Anatomically, it presents itself in the form of reddish or gray patches, of varying consistence, but often hard, and sometimes slightly shrunken. Microscopically, it consists mostly of an interstitial inflammation, resulting in a fibrillated or amorphous tissue, with numerous nucleated cells, oval, fusiform, or stellate. The nerve-fibres are generally destroyed, and in the gray matter the nerve-cells may also disappear, or be reduced to small angular bodies. The arteries are thickened, and the interstitial tissue is often especially abundant around them. In recent stages, granule corpuscles may be found.

When the lesion is situate in a conducting path, secondary degenerations take place upwards or downwards, according as a motor or sensory path is involved.

Symptoms.—These vary, in different cases, with the localisation of the lesions, and resemble those of the acute forms. A transverse lesion causes paralysis, with some anæsthesia, often very little, the whole developing in the course of months or years; ultimately excessive reflexes and spastic rigidity supervene. The bladder also is generally affected. If the two sides of the cord are affected unequally, one leg is more paralysed than the other, or even a unilateral lesion may exist with paralysis in one leg and anæsthesia in the other. If the cervical or lumbar region is affected, paralysis and anæsthesia may be accompanied with muscular atrophy, from implication of the nerve-centres of the brachial and lumbar plexuses respectively; and with this there will be some loss of electrical irritability, or even the degenerative reaction may occur.

Diagnosis.—Many cases are distinguished by the irregular way in which the symptoms are grouped. A localised transverse myelitis may be confounded with compression by tumour or caries, or with primary spastic paraplegia. In compression there is generally more evidence of irritation, and other symptoms due to tumour or spinal disease may be detected. Primary spastic paraplegia is distinguished by the absence of sensory symptoms, though the motor conditions may be closely similar in the two diseases. More diffused forms may resemble pachymeningitis or progressive muscular atrophy: in the former there is more anæsthesia, and often more pain in the back; in the latter, sensory symptoms are absent.

Treatment.—The disease is little amenable to treatment, but is sometimes arrested. The most efficient means are rest, change of air, tonics, and the use of counter-irritation by mustard plasters, blisters, or even the actual cautery.

Brown-Séquard recommended a hot douche to the back at a temperature of 100° to 104° F. Gowers recommends, as drugs, arsenic, small doses of the red iodide of mercury ($\frac{1}{24}$th grain), and iodide of iron; and is of opinion that neither mercury in large doses nor potassium iodide does any good. The same complications will have to be treated as in acute myelitis.

LANDRY'S PARALYSIS.

(*Paralysis Ascendens Acuta.*)

In 1859 Landry described some cases characterised by a paralysis commencing in the lower extremities and rapidly ascending; and for this no pathological cause could be found in the instances, which ended fatally. Several other cases of similar nature have since been recorded.

Ætiology.—The disease affects males more than females, and is most frequent between the ages of twenty and forty. It has occurred after exposure to cold, and in convalescence from acute diseases, in patients addicted to alcohol, and after syphilis.

Symptoms.—In some cases there are premonitory symptoms, such as malaise, pain in the head and back, and numbness and tingling, but usually the disease begins with weakness in the legs, often one before the other. This soon increases to marked paralysis, and invades successively, and within a few days, the thighs, trunk, abdomen, and arms; and these, like the legs, are not always affected simultaneously. The diaphragm, and the muscles of the neck of the palate, and those subserving articulation are subsequently paralysed. Very rarely other cranial nerves are affected: thus, diplopia, paralysis of accommodation, dilatation of one pupil, and facial paralysis have been noticed. The sensory functions are much less profoundly disturbed; but there may be, besides subjective sensations, slight anæsthesia, or hyperæsthesia, or tenderness of the muscles. In rapidly fatal cases the muscles have not wasted, and the electrical reactions have appeared to be normal; but in some cases of longer duration both atrophy of muscles and modifications in electrical properties have been observed. The sphincters are generally active, there is no tendency to bed-sores, the cerebral functions are perfect, and there is no fever, except in a few cases at the very onset. The knee-jerks are always absent, and the cutaneous reflexes in most cases.

The mortality is high—*e.g.*, 58 per cent. in cases collected by Ross. The duration of the disease is from two days to two or three weeks in fatal cases, and death occurs mostly from paralysis of the diaphragm and intercostal muscles. On the other hand, the symptoms persist from two to six or seven months in cases which recover.

Pathology.—In some cases the spinal cord, nerves, and muscles have been found completely free from disease; in others, minor or quite localised changes have been found in the spinal cord; in others, again, inflammatory changes in the nerves, or neuritis. The spleen has occasionally been found enlarged, and micro-organisms are said to have been discovered in the nerves.

The nature of these cases is still open to question. While multiple neuritis was practically unknown, Landry's paralysis was regarded as arising from a disturbance of the motor functions of the spinal cord, and as possibly due to some toxic cause. It must now be admitted that there are many variations in the cases, that the sensory, electrical, and nutritive functions are not always entirely exempt, and that there is, in many cases, a close resemblance to multiple neuritis. The name acute ascending paralysis is not distinctive as regards the pathological cause, and it seems probable that some of the cases are instances of an acute diffuse (toxic) myelitis; others of a severe peripheral neuritis; others again, perhaps, of a toxic influence upon the cord chiefly involving the motor neurons.

Treatment.—This may be the same as that of multiple neuritis.

ANTERIOR POLIOMYELITIS.

This disease has received several names. The acute cases were formerly called *infantile paralysis*, from their very great frequency in young children; and *essential paralysis*, from ignorance of their origin in disease of the spinal cord. It has, however, been clearly shown that the lesion is an acute inflammation of the anterior cornua, and it has been accordingly called *anterior cornual myelitis*, or *poliomyelitis anterior acuta* (from πολιὸς, grey). The term *atrophic spinal paralysis* refers to its effect upon the muscles. More recently the occurrence in adults of a disorder precisely similar to that of children has been recognised, and it is clearly not desirable that the terms *infantile paralysis* and *adult spinal paralysis* should be used side by side without their identity being indicated. The pathological names *poliomyelitis* and *cornual myelitis* are more distinctive than any of the clinical titles.

ACUTE ANTERIOR POLIOMYELITIS.

(*Infantile Paralysis.*)

Ætiology.—The most striking feature is the frequency with which it affects young children; it occurs six times more often in the first ten years than in the remainder of life. It has been attributed to cold and to falls; but there are many facts in favour of its being of an infective nature. It has occurred during convalescence from acute diseases, immediately after attacks believed to be influenza, and in women during the puerperal state. In a case under my care it began actually during confinement. Further, epidemics have been observed affecting a family or larger numbers.

Symptoms.—The onset of the illness is often sudden, but the paralysis is not generally noticed for one, two, or three days. The early symptoms consist of feverishness, or convulsions, or severe pains, either general or localised to the limb or limbs that are afterwards paralysed.

Both pains and feverishness are often present together, and the pain may persist some days. The way in which the paralysis itself begins is also very variable; it may show itself in one limb, and within two or three days affect others; on the other hand, sometimes three or four limbs are paralysed at first, and recovery rapidly takes place in one or two, leaving the others permanently affected; finally, in other cases, certain limbs are affected from the first and remain so. A child, aged four and a half, was sick one day, feverish with headache the next two days, and became paralysed in all four limbs on the fourth. Three months later paralysis of both arms and slightly of the right leg remained. An adult had acute pains all over him, with general and severe prostration; after thirty-six hours he was paralysed in the lower extremities, the right being less affected than the left. A child, aged fifteen months, was taken rather suddenly ill one day, and appeared to have something wrong with the hip. She was feverish for three days, then the left leg was found to be painful and paralysed. The pain subsided in ten days, but the paralysis persisted.

I have hitherto spoken of limbs being affected, but it is characteristic of the disease that it does not necessarily affect the whole of a limb, but often only a part, or even one muscle: thus the upper arm, or the forearm, the anterior tibial muscles, the muscles of the thigh, the deltoid, some muscles of one limb with some of another, or the whole of one limb with part of another may be paralysed; and even if paralysis affects the two legs together, or the arm and leg on one side, it generally wants the uniform distribution of ordinary paraplegia or hemiplegia. The affected muscles rapidly undergo atrophy, lose bulk, and become flaccid; and the electrical conditions are changed. The faradic current, whether applied to nerve or muscle, excites no contraction, the galvanic irritability may be at first increased, and polar changes are observed, thus constituting the complete reaction of degeneration. In severe cases galvanic reactions ultimately disappear. The reflexes are quite lost, knee-jerk disappearing when the lower extremities are involved. Sensation is never seriously affected; though there may be pain, tingling or formication in the early stages. The bladder and rectum are always unaffected. After the first few days there is no further change for the worse in the extent or distribution of the paralysis. No fresh paralysis occurs, and any improvement of the affected muscles takes place with extreme slowness. According to the number of muscles atrophied will the use of the limbs be impaired; but after a time, in many cases, lost movements are restored by fresh combinations among the muscles that have been spared. Atrophy is, in almost all cases, a prominent feature, hollowing out the rounded part of the forearm, or reducing the upper arm or the leg to a mere stick. Sometimes, however, the loss of muscle may be entirely concealed by the presence of fat; the flabby condition of the muscle even then can be generally recognised. Associated with the atrophic condition of the muscles is generally a change in the vascularity of the limb; it is cold, shrunken, and blue or livid from retarded circulation. The nutrition of the bones and other parts is also involved, so that a limb paralysed in infancy or early childhood does not grow with the same rapidity as its fellow, and may be shorter by half an inch, an inch, or more. Lastly, deformities occur besides those directly due to loss of muscular substance. Some are the simple result of failing muscular support; thus, from atrophy of the deltoid, the humerus falls from the glenoid cavity. Others consist of permanent changes in the position of the limbs, such as talipes equinus, which so often results from paralysis of the anterior tibial muscles. This was long attributed to the unresisted contraction of the calf muscles, but it has been shown by Volckmann that the chief element in its causation is the weight of the foot, which, whether the patient is upright or recumbent, falls into a position of extension unless supported by the anterior tibial muscles. The position thus constantly assumed becomes fixed by connective-tissue changes, both in the shortened calf muscles and about the ankle-joint.

The disease itself is rarely, if ever, fatal; though it is conceivable that such a result should be brought about if the lesion extended to the centres of the medulla oblongata.

Cases that have been investigated pathologically within a few months or years of the beginning have mostly died from other diseases, such as pneumonia, or the exanthems. On the other hand, complete recovery is rare. Nearly always one or more of the affected muscles remain atrophied. Improvement in locomotion, or the use of the arms, may go on for several months, but even then it is obvious, from the outline of the limb, a limping gait, much pelvic movement, or a swinging foot, that a great deal remains uncured.

Morbid Anatomy.—Several cases have been examined after one or two years' duration; but quite early cases are less numerous. Drummond found in a case, presumably of this nature, which was fatal in a few hours, redness of the anterior grey cornua, arterioles and capillaries distended with blood, minute extravasations of blood, and swelling of the neuroglial elements and of the ganglion-cells. In cases of five or six weeks' duration, vascular distension, minute hæmorrhages, disappearance of the ganglion-cells, and degeneration of the anterior roots have been observed (F. C. Turner, Williamson).

In later stages, the cord presents changes obvious to the naked eye: the motor nerve-roots, coming from the part presumably affected, are diminished in size and number. On a transverse section, the cord is smaller on the affected side, and the anterior cornu is shrunken. Under the microscope, there is an almost entire absence of motor nerve-cells and axis-cylinders; the few nerve-cells that remain are smaller than normal, shrunken, fusiform, and wanting in processes. They lie in a dense connective tissue, which may be almost felt-like in structure. The motor nerve-roots, both in and beyond the cord, show the destruction of axis-cylinders, and are obviously degenerated. Thus, the lesion is seen to be mainly an acute inflammation of the anterior cornu, followed by sclerotic changes, involving the destruction of lower motor neurons with the necessary results in nerve-fibres and muscles. The last are pale pink, watery in appearance, and present under the microscope the changes described as the result of lesions of motor nerves (p. 177). Gowers suggests that in some cases a hæmorrhage may be the primary lesion.

Diagnosis.—In the early stages it is possible to mistake it for other febrile affections. The pain that is sometimes present may suggest *rheumatism*, but it is situate rather in bone and muscle than in the joints. When the paralysis develops, within two or three days the diagnosis is generally clear, and it is confirmed by the rapid atrophy, the loss of reaction to the faradic current, and the changed reaction to galvanism. In cases of old standing, while the muscular atrophy, with retained sensation, indicates a lesion of the anterior cornu, the history of acute onset distinguishes the lesion from others less acute, such as *progressive muscular atrophy*. It is important not to be induced, by the term "infantile paralysis," to think that other forms of paralysis never occur at an early age. Paraplegia may result from spinal disease, or a tumour of the cord; and hemiplegia occurs from embolism, or congenital lesions of the brain. In these cases there is not the same rapid atrophy of muscles, and the electrical reactions are normal because in them the upper motor neuron is chiefly involved, and not the lower.

Treatment.—In the earliest stage, before the paralysis has developed, attempts may be made to reduce the fever, but the treatment must be mainly expectant. When the paralysis has declared itself, the patient, already in bed, may be placed in the prone or lateral position, and ice-bags may be applied to the spine. Ergotin, in doses of $\frac{1}{6}$ to $\frac{1}{2}$ grain for children, and 1 or 2 grains for adults, twice daily, and belladonna, in doses of $\frac{1}{20}$ to $\frac{1}{6}$ grain of the extract, according to the age of the child, have been recommended. After a month or so, the local treatment of the atrophied muscles becomes the most important. Electricity, in the form of faradism, to those muscles which are still susceptible to it, and galvanism to others, should be employed. Massage is also of value in promoting the circulation of the limb.

Every effort, also, must be made, by position and passive movement, to prevent deformities in the affected limbs. Mechanical supports, and in some cases the division of tendons, may be necessary. The patient will be generally benefited, after the first few weeks, by the use of tonics, such as iron iodide and phosphate, small doses of arsenic or strychnia, and cod-liver oil.

Chronic Anterior Poliomyelitis.

(*Poliomyelitis Anterior Chronica.*)

Subacute and *chronic* forms of atrophic spinal paralysis have been described; but it seems that some of these must have been instances of peripheral neuritis. True subacute cornual myelitis occurs in adults more often than in children. The legs are first affected with paralysis, and afterwards atrophy; but instead of the symptoms reaching their maximum in a short time, the atrophy progressively increases for months or a year or two. It may then become stationary, and ultimately much improvement or recovery may follow; but in some cases death results from exhaustion or respiratory paralysis. In the altered electrical reactions, on the one hand, and in the normal condition of the sensory functions, and of the bladder and of the rectum, on the other, it resembles the acute form of the disease. It commonly affects the lower extremities first, and only after a considerable period attacks the arms.

HÆMORRHAGE INTO THE SPINAL CORD.

This is a very rare occurrence, and contrasts remarkably with hæmorrhage into the brain, which is one of the most common causes of cerebral paralysis.

Ætiology.—It occurs in younger persons than does cerebral hæmorrhage; and in males more often than in females. The chief causes are, (1) alterations in the vascularity of the cord, or structural changes in the walls of its blood-vessels; (2) a preceding lesion of the cord, such as a soft gliomatous growth, the vessels of which may rupture, or, perhaps more often, an acute myelitis in its early stage; (3) injuries, falls upon the feet, strains, &c. which form the most frequent exciting cause. A primary hæmorrhage is generally confined to the grey matter, and is of small extent, rarely exceeding the size of an almond; but in cases of congestion it is punctiform, and may occupy both white and grey matter. It is also more diffused when secondary to myelitis.

Symptoms.—The onset is often quite sudden: the patient may be seized with acute pain in the back, and then fall, with complete paralysis of motion and sensation below the seat of the lesion. In other cases the symptoms may be more gradually developed in the course of a few hours. The limbs are mostly relaxed, and there may be clonic contraction in the muscles, either immediately or in a few days. The symptoms are subsequently those of an acute local myelitis—paralysis, loss of sensation, the reflexes increased after a short period during which they are diminished, and the bladder affected. There may be elevation of temperature after a few days from secondary inflammation, and this myelitis may spread upwards and downwards. Secondary degeneration of the lateral and posterior columns frequently follows, and accompanying this is spastic rigidity of the limbs, with increased knee-jerk and ankle-clonus; but if the grey matter is much destroyed in the cervical or lumbar regions, wasting of the corresponding muscles may supervene. Trophic changes, cystitis, and bed-sores are also not infrequent.

Diagnosis.—This depends on the sudden onset of the symptoms, but the disease may be confounded with a *hæmorrhagic myelitis* and with *meningeal hæmorrhage.* Prodromal symptoms of even very short duration, and fever, make myelitis probable. *Meningeal* or *extra-medullary hæmorrhage* is distinguished by the signs of nerve-irritation rather than nerve-impairment, such as severe pains in the distribution of certain nerves, and muscular cramps, as compared with anæsthesia and paralysis in spinal hæmorrhage. The bladder is less likely to be affected, bed-sores do not occur, and the disease is less fatal. *Acute anterior poliomyelitis* begins as an acute myelitis, and it is even possible that there is in some cases hæmorrhage, but the symptoms are less sudden. The absence of back-pain, the initial fever, or convulsion, the freedom from vesical, rectal, and sensory symptoms, and the rapid localised atrophy readily distinguish this disorder from spinal hæmorrhage.

The **Prognosis** is unfavourable; many cases are fatal; and others develop into conditions of permanent chronic paralysis, like cases of myelitis. In some instances there is rapid recovery up to a certain point, with no further improvement.

Treatment.—The patient should be placed in the prone position, or on the side if possible, to prevent the spinal cord being in the lowest part of the body. Ice-bags should be applied to the spine; and if symptoms are progressing, blood may be withdrawn by leeches or cupping to the back, or leeches to the anus. Ergotin should be injected in doses of three grains, every two or three hours, up to three or four doses, or the liquid extract of ergot may be given by the mouth. Pain may require to be relieved by sedatives. Later on the treatment is similar to that of myelitis.

DIVERS' PARALYSIS.

Divers are liable to a form of paraplegia, which supervenes on their return to the surface from great depths, such as are equivalent to a pressure of three or more atmospheres; and this is more likely to occur if they are brought up quickly than otherwise. Workers in caissons are liable to the same paralysis under similar circumstances. The symptoms may be mild or severe, and come on within an hour of the changed conditions. In mild cases there is only a little weakness with numbness of the lower extremities, which passes off in a few hours or days; in other cases the arms are also affected, or the symptoms are more severe and last several weeks; in others again there is coma and the patient may die in it.

The explanation appears to be that in consequence of the pressure an excessive quantity of gas is forced into the blood, and that on return to the upper air, the gas rapidly escapes from the blood, perhaps even from the vessels, and presses upon the tissues of the spinal cord. In one case fissures were found in the cord, filled with leucocytes; hæmorrhages have been found, but are not constant.

Most cases recover, and in mild cases little treatment is required; more severe cases should be dealt with as cases of acute myelitis.

LOCOMOTOR ATAXY.

(*Tabes Dorsalis.*)

Locomotor ataxy is a very chronic disease, characterised, in its developed form, by inco-ordination of movements in walking, although the muscles retain the power of contraction; and dependent mainly upon a chronic degeneration with sclerosis of the posterior columns of the spinal cord (posterior sclerosis).

It has been included with some allied diseases (Friedreich's ataxia, spastic paraplegia) in a group of system diseases (*Germ.* System-Krankheiten) distinguished by the separate affection of certain tracts of fibres, or neuron-systems; but locomotor ataxy represents the group badly because its lesions are wide spread and by no means limited.

Ætiology.—It is much more frequent in males than females, in the proportion of ten to one. It affects mainly the middle period of life, between twenty and fifty; and very rarely occurs before or after that period.

The constancy with which syphilis occurs as an antecedent makes it practically certain that it is the chief cause. The lesions are not of a gummatous or tertiary syphilitic nature, but consist of degenerations of nerve elements, probably brought about by a substance of the nature of a toxin. Cold and wet, concussions and injuries of the spine, and sexual excesses may assist by depressing the general vitality.

Symptoms.—Although the name commonly employed refers to the failure of only one function, there is scarcely a disease in which the functional nervous disorders are so numerous and widespread; with the result that in different cases different symptoms may be more prominent, and lead to errors in diagnosis unless their connection with the spinal lesion is familiarly known.

In the early (pre-ataxic) stage the characteristic symptoms are pains in the limbs, loss of knee-jerk, and loss of pupil light-reflex. This stage may last for months or years.

The pains are known as *lightning pains;* they are severe shooting, stabbing, or darting pains in the lower extremities, sometimes resembling electric shocks. They are often looked upon as rheumatic, but they are seated in the muscles and bones, and not in the joints. They come on suddenly, and it may be with such severity as to make the patient start up in bed, or cry out. They may subside in a few minutes, but generally soon recur, and continue thus, coming and going, for several hours. They may then disappear, and not return till the next day, or after an interval of days or weeks. They thus present the greatest irregularity both as to recurrence and duration, The *knee-jerk is abolished* quite early, and this is one of the earliest and most constant symptoms of locomotor ataxy. The plantar reflex is sometimes absent, at others present or even exaggerated.

In four-fifths of the cases, the pupil fails to contract to the stimulus of light, though it continues to contract during accommodation for near vision (*Argyll-Robertson pupil*). In addition to this, there is often, quite early, slight *anæsthesia* of the feet and lower part of the legs, and occasionally temporary paralysis of one or more of the ocular muscles, leading to diplopia, or squinting, or ptosis, according to the muscle involved.

In the second stage, or the stage of the developed disease, the prominent feature is the *muscular inco-ordination* of the lower extremities, and this is associated with increased anæsthesia and other sensory disorders, and impairment of the functions of the bladder.

Other rarer conditions, which are generally first observed in the early stage, are the so-called gastric and other *crises;* certain trophic disturbances; optic nerve-atrophy, myosis, and other ocular conditions.

The *muscular inco-ordination*, or *ataxy*, as indicated by the epithet "locomotor," is chiefly and first noticed in the lower extremities, and is confined to them in a great number of cases. At first the patient is only slightly unsteady in his gait, finds a difficulty in walking quite straight, separates the legs a little to meet this difficulty, keeps his eyes carefully fixed on the path he is walking, and readily loses his balance when trying to turn. In the dark, when the guiding sensations of sight are removed, he is still more unsteady. If he shuts his eyes also, he cannot even stand when his feet are placed close together, so as to reduce the base of support: he then sways to and fro, or from side to side, and soon loses his balance altogether (Romberg's sign). In a later stage, walking can be still accomplished, but the legs are drawn up or jerked up in a disorderly way; they are often thrown violently forwards, and the heels are brought down with force upon the ground. Turning is still more difficult than before, and has to be effected with great care and the assistance of a stick, a wall, or a friend. Movements may also take place when the patient is quiet (*static ataxy*). Nevertheless, the muscular power remains good. The patient can bear another man upon his back, and if he sits in a chair he can keep his leg extended in front of him against any ordinary attempts of the medical man to flex it. Moreover, the muscles are of normal bulk, and give the normal electrical reactions. The distance which the patient can walk is lessened to a mile or two, on account of the great waste of strength involved in these disorderly and, therefore, ineffective movements. In later stages, the ataxy may be such that the patient cannot get about at all without assistance from sticks, a chair, or a friend on either side; and, finally, he may have to take to his bed.

The upper extremities are sometimes affected late in the disease, but the ataxy is generally less extensive than that observed in the legs.

Anæsthesia is variable. It affects the feet and legs, spreading to the knees, or even to the thighs and buttocks, and sometimes to the trunk. If the upper extremities are affected, it begins in the fingers and hands, creeping gradually up the forearms. The loss of sensation gives rise, when the patient is standing or sitting, to a peculiar feeling of being on some soft substance, which patients describe as being like water, wool, sponge, or india-rubber. Numerous other modifications of sensation are observed in different cases—burning or gnawing pains in the extremities, more continuous than the lightning pains; a sense of constriction in the legs, groins, genitals, or trunk, the latter often described as "girdle-pain"; tingling, pins and needles, sensations of cold or heat, and increased sensitiveness to alterations of temperature.

The anæsthesia may take the form of diminished sensibility to pain alone, or to touch alone, or differences of temperature may be less readily perceived. In some cases the perception of pain or of heat and cold is delayed, or the pain recurs after the source of it has been removed, or the allochiria or polyæsthesia above described (p. 168) is present. The deeper tissues are also anæsthetic, certainly the muscles, and probably the joints, fibrous tissues, and tendons. The *loss of the muscular sense* is an important part of locomotor ataxy, and may be shown by the patient's inco-ordinate movement, and ignorance of the position of his limbs, when his eyes are shut.

The *bladder* is often affected as follows: In early stages, there is irritation, with frequent micturition, and the necessity of passing urine directly the desire is perceived. Later on, the detrusor is weakened, and the urine comes in a sluggish stream, or merely dribbles away. Sometimes there is retention with incontinence from overflow. The sphincter ani is generally weakened, or fæces are passed unconsciously from insensibility of the anus. Sexual power is commonly lost.

Certain curious functional disturbances of the viscera have been called *crises.* The best known are the gastric *crises* (*crises gastriques*). They consist of attacks of severe pain in the epigastrium, passing through to the back, or extending from the groins up to the shoulders, accompanied by vomiting, at first of clear liquid, later of bile, and even of blood. Pain may occur without vomiting, or vomiting without pain. Often, also, there is palpitation or irregularity of the heart. These symptoms last for two or three days, and then subside, leaving the functions of the stomach quite normal. The other *crises* that have been described are *rectal* crises, consisting of paroxysmal pain in the rectum, with severe tenesmus; paroxysms of renal pain, or *renal crises;* pain in the bladder or urethra—*vesical* or *urethral crises; laryngeal crises*, consisting of laryngeal spasm, with inspiratory and expiratory stridor, cough, and dyspnœa; and *bronchial crises*, when there are paroxysms of coughing. Paroxysmal diarrhœa has been thought to represent an *intestinal crisis.*

The *trophic* disturbances which occasionally occur in locomotor ataxy are œdema of the feet, local sweating, ecchymoses under the skin, brittleness of the hair, and herpes, the last three in connection with severe pains. The skin of the sole of the foot becomes thickened, or blisters, or may present a deep circular and conical depression, the "perforating ulcer." The nails become thickened and furrowed, or fall off, and are slowly renewed. Teeth decay, or may all fall out within a short time.

In occasional cases, important changes take place in the *bones* and *joints.* The bones become brittle; the compact tissue has been found thinner and more porous; fractures occur spontaneously, or with the slightest amount of force; and a great deal of callus forms in the process of union. The joint-changes in locomotor ataxy have formed an interesting subject of discussion the last few years, under the name of *Charcot's disease of the joints.* The changes which take place are almost identical with those which occur in osteo-arthritis, or rheumatoid arthritis, namely, erosion of cartilage, wasting of the head of the bones, ossification of the ligaments, and new bony outgrowths. Clinically, they are characterised by rapid painless swelling from effusion into the joint, and subsequently extreme mobility and grating. While some hold that these changes are the direct result of the withdrawal of trophic influence from the part, others consider them to be due to the external injuries, strains, &c., which ataxic limbs are so much more likely to suffer than healthy ones.

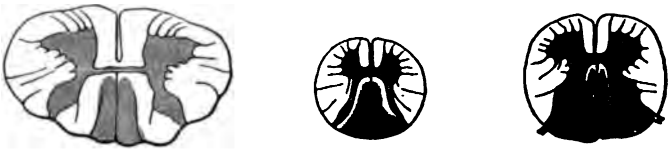
In addition to the loss of light-reflex the pupils may show other disturbances, such as inequality, extreme contraction, failure to dilate on pinching of the skin, or failure to contract with accommodation. Primary (grey) atrophy of the optic nerve occurs in about one-tenth of the cases, starting on the temporal side; this results in contraction of the visual field from the periphery inwards, and loss of vision for colours (*dyschromatopia*) in the following order: green, red, yellow, blue, and violet.

Deafness, paralysis of the abductors of the vocal cords, severe headache, and apoplectiform or epileptiform attacks occasionally occur.

Course of the Disease.—Though spoken of as progressive, the symptoms are often stationary for very long periods, and the disease may last twenty years or more. Especially the pre-ataxic stage may persist for several years; and even sufferers who are unable to leave their beds may live to old age. Cases with early optic atrophy often progress slowly. Thus, the majority of patients die, not from the disease itself, but from intercurrent affections, such as pneumonia, phthisis, bronchitis, or other independent ailment. Cystitis and renal complications, bed-sores and pyæmia, and rarely laryngeal spasm, are the more direct consequences of the disease, which may be fatal. Some cases terminate in general paralysis of the insane.

Morbid Anatomy.—The change constantly found in the spinal cord is a degeneration of nerve-fibres and sclerosis of neuroglia in the posterior columns. It is seen as a grey discoloration of the white matter, and is brought out more readily by the hardening and staining processes. In ordinary cases it occupies in the lumbar region the whole of the posterior columns, but above the lumbar region the sclerosis of the postero-external columns ceases, whereas the sclerosis of the posterior median columns continues up into the cervical region. The lesion of the postero-external column is most intense in its posterior portion, the anterior portion near the commissure being sometimes free. In very severe cases involving the arms, the postero-external columns may be sclerosed in the higher parts of the spinal cord as well as in the lumbar region. Lissauer's tracts are said to be early affected; and the antero-lateral ascending tract and the cerebellar tract are occasionally degenerated. In some cases changes may also be detected in the grey matter; they consist of atrophy and degeneration of nerve-cells or fibres in the posterior horns, in Clarke's columns, in the tractus intermedio-lateralis, and even in the anterior cornua. Under the microscope, degeneration and disappearance of the nerve-fibres, increase of the connective tissue, which is fibrillated, and thickening of the walls of the arteries are observed. The pia mater is often thickened over the posterior columns, or even over the lateral columns as well, or completely round the cord.

FIG. 14.



Cervical. **Dorsal.** **Lumbar.**

Position of Lesions of Locomotor Ataxy in different parts of the Spinal Cord.

The posterior nerve-roots are generally atrophied down to the spinal ganglia, which are generally healthy as well as the mixed nerves beyond them; but atrophy of the peripheral nerves has been also found, mostly of those supplying the skin and the joints, and in the legs more commonly than in the arms. In the optic nerves, in cases alluded to, are found atrophied nerve-fibres, and increased connective tissue. The nuclei of atrophied ocular muscles are degenerated; but the lesion of reflex iridoplegia has not yet been found.

Pathology.—It is clear that the lesion of tabes dorsalis is one which begins below and ascends the cord; and that the sclerosis of the posterior median columns is secondary or consecutive to that of the postero-external columns below. There is therefore a primary lesion in the lower sensory neurons; and other sensory neurons may be also affected, such as those of the cerebellar tracts, and the optic nerve. Thus are explained some early features of locomotor ataxia, *e.g.*, the pains, anæsthesia, and lost reflexes. Inco-ordination of muscular movement, though at first sight a motor derangement, may clearly be brought out by interference with the paths by which sensory impressions are conveyed to the centre. But it has long been shown that ataxy does not depend necessarily on cutaneous anæsthesia, since either may exist without the other; and Gowers gives good reasons for regarding this symptom as depending on a lesion of the muscle-sense—that is, of the sensory nerves proceeding from the muscles, and passing by the nerve-trunks and posterior roots, probably then by the posterior median column and cerebellar tract, to the cerebellum. Since the muscle-sensory nerves, equally with the cutaneous nerves, are contained in peripheral nerve-trunks, and the posterior roots, it might be expected that ataxy would sometimes result from lesions of these parts; and, as a fact, peripheral neuritis from alcoholism may cause ataxy, and all the symptoms of tabes dorsalis have been seen in a case of multiple tumours of the posterior roots recorded by Hughes-Bennett. In ordinary locomotor ataxy some part of the inco-ordination may be due to the peripheral lesions, to which also the trophic changes must be referred.

Diagnosis.—Locomotor ataxy has to be recognised in its early stages before inco-ordination is pronounced; and it has to be distinguished in its second stage from other disorders affecting the power of the lower extremities. The lightning pains are generally very characteristic, but the absence of knee-jerk and the loss of light-reflex of the pupil are the distinctive features. In a more advanced case the same two signs are of service, and in addition, the inability to stand with the eyes shut, or to turn with steadiness. Where locomotion is much interfered with, the case contrasts with *paraplegia* from myelitis, by the retention of absolute muscular power, and by the normal bulk of the muscles, with absence of rigidity. Where muscular wasting and weakness supervene there may be more difficulty in diagnosis, but the long history and the course of the symptoms will assist. *Cerebellar* disease also causes ataxia, but it is generally of a reeling, staggering kind, the patient swaying from side to side, falling over, crossing the legs to recover balance, and presenting a close resemblance to a drunken man; whereas, in locomotor ataxy, for a time, the gait may be steady in direction, but the feet are jerked forward, and the heel or flat of the foot is brought down sharply on the ground. The two diseases have also their special accompanying symptoms.

It is important to remember that a patient with gastric crises, a perforating ulcer of the foot, or "Charcot's joint," may be entirely unaware of any locomotor symptoms, and in such cases the knee-jerk and the pupils should be at once tested.

Peripheral neuritis in its ataxic form may be generally recognised by the atrophy and tenderness of the muscles, the "dropped foot," the high-stepping gait, the altered electrical reactions, the normal pupils, and possibly by an alcoholic history.

There are two forms of ataxy, to be described shortly, which differ in certain points from the usual posterior sclerosis—namely, ataxic paraplegia, and hereditary ataxy, or Friedreich's disease. It will be shown later that general paralysis of the insane occasionally commences with symptoms like those of locomotor ataxy; the two diseases are probably closely allied.

Treatment.—This is by no means satisfactory; yet it is not uncommon for patients to be relieved of certain symptoms after a few months. Thus, pains disappears, anæsthesia diminishes, and incontinence of urine becomes less under treatment, although other features of the illness remain in full force.

A number of drugs have been given in locomotor ataxy; nitrate and phosphate of silver, arsenic, strychnia, iron, quinine, belladonna, ergot, phosphorus, Calabar bean, iodide of potassium, and mercurials. In spite of its frequent occurrence in syphilitic subjects, the last two drugs are rarely of any value. The silver salts must be given with caution, in ¼ or ½ grain doses, for not more than two months at a time, for fear of producing a deposit of metal in the skin (argyria). Gowers speaks highly of arsenic. Counter-irritation to the spine may be used in recent or rapidly developed cases. Galvanism, in the form of the continuous current to the spine and legs, has often seemed to me to do good to the extent above indicated. For the pains morphia may require to be given, but should be avoided as long as possible. Cannabis indica is sometimes effectual, in doses of ¼ or ½ grain every three or four hours during an attack of the pains. Antipyrin has been tried with success, in doses of 15 grains every two or four hours. Acetanilide, methylacetanilide, or phenacetin may be also given.

Attention to the bladder is very necessary, and the catheter should be used if any urine is retained. The expulsive power may be increased by strychnia, or incontinence may be lessened by belladonna. Gastric attacks may require morphia, or may be checked by blisters to the epigastrium. For the laryngeal spasm, Gowers recommends amyl nitrite and the local application of cocaine. Stretching the sciatic and other nerves has been tried rather freely in locomotor ataxy, but without sufficient success to justify its continued use. Nor has the suspension of the patient by his head and shoulders, with the object, probably never attained, of stretching his spinal cord, been more fruitful of good results.

PRIMARY SPASTIC PARAPLEGIA.

(*Primary Lateral Sclerosis.*)

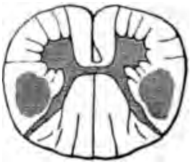
The term *spastic paraplegia* is applied to cases in which weakness of the limbs is associated with chronic spasm of the muscles, so that the limbs are constantly rigid. The muscles are usually at the same time well nourished, and the reflexes increased. This condition generally occurs as a sequel of cases of paraplegia, due to a transverse lesion of the spinal cord, which leaves the lower part of the cord intact, and is associated with, and possibly due to, the descending degeneration or sclerosis which affects the posterior part of the lateral columns, and to a slight extent the inner margin of the anterior columns—that is, the crossed and direct pyramidal tracts or upper motor neurons (*see* p. 210). The spastic condition is in those cases ascribed to a *lateral sclerosis*, which, coming as a result of a local disease of the cord (myelitis or compression), must be called *secondary*. On the other hand, cases come under observation, from time to time, in which a spastic paraplegia has developed without anything to indicate that it has been preceded by a localised lesion of the spinal cord. These cases have been ascribed to a primary degeneration of the neurons in the pyramidal tracts, which results like the lesion of tabes dorsalis in a thickening of the neuroglia or sclerosis, *i.e.*, a *primary lateral sclerosis.* The pathological proof of this is as yet incomplete, for no case of uncomplicated sclerosis of the lateral columns has been observed *post mortem*, but the combination of lateral sclerosis with disease of the anterior cornua, in cases of muscular atrophy with rigidity (amyotrophic lateral sclerosis), and with posterior sclerosis in ataxic paraplegia (spastic ataxia), strongly supports the connection in question.

Ætiology.—This disease is more common in males than in females, and occurs mostly between the ages of twenty and forty.

It has occasionally seemed to be due to cold or wet, or a fall; often no cause is apparent. Syphilis has no special connection with it, as it has with locomotor ataxy.

Symptoms.—The disease develops very slowly and insidiously. It begins with weakness and stiffness of the lower extremities; the legs feel heavy, the patient soon gets tired in walking, and, as time goes on, the distance he can walk without fatigue gets less and less. The knee-jerk is excessive; ankle-clonus is present, and the cutaneous reflexes are usually increased. After a time he has to help himself with sticks; the legs are rigidly extended and firmly adducted, and it is almost impossible to get one in front of the other. The rigidity is nearly constant, or, if the muscles relax slightly, they at once contract on a touch, or the stimulus of attempted movement. The arms are generally unaffected, but if they are involved they take up a position of rigid flexion. In some cases the muscles of the trunk are involved. The muscles mostly remain in good condition, and the electrical reactions are normal. Sometimes, however, the excitability of the muscles to electricity is slightly increased, at others diminished, to both currents. Sensation is unaffected beyond the occurrence of rheumatoid pains and some tingling or numbness; and the bladder and rectum are in most cases free from the functional disturbances common in paraplegia and locomotor ataxy. The course of the disease is very chronic, and it is less threatening to life than are other forms of chronic spinal disease. It may be complicated in its late stages by extension to the anterior cornua, causing wasting of the muscles with loss of reflexes, or to the postero-external column, producing lightning pains and ataxy.

FIG. 15.



Position of Lesion in Primary Spastic Paraplegia.

Diagnosis.—The recognition of a spastic paraplegia is not difficult; the important point to be decided is whether the condition is primary or secondary, and very careful inquiries and examination should be made before concluding that a case of rigidity is not secondary to local disease of the spinal cord, such as transverse myelitis or compression. In primary disease of the lateral tracts, weakness and stiffness come on gradually and simultaneously; whereas in secondary cases there is at first decided paralysis, rigidity only supervenes later, and sensory symptoms and girdle pain may be present. The same care will enable one to recognise the cases in which lateral sclerosis is combined with disease of other parts of the cord: for instance, with disease of the anterior cornua (amyotrophic lateral sclerosis); with disease of the posterior columns (ataxic paraplegia); or with disseminated sclerosis, in which case the lateral sclerosis may be partly secondary to patches of disease seated in the pyramidal tracts. Lateral sclerosis may also form a part of general paralysis of the insane.

A spastic paraplegia occurring in young children or infants is generally of cerebral origin (*see* Infantile Spastic Paraplegia).

Treatment by drugs is unsatisfactory. Arsenic, nitrate of silver, ergot, hydrobromic acid, potassium bromide and potassium iodide have been given. Rest is very desirable. Friction of the muscles, combined with the hot bath or Turkish bath, sometimes relieves the spasm. Electricity is of doubtful value; and, in general, it is desirable to avoid undue stimulation of the muscles in any way.

ATAXIC PARAPLEGIA.

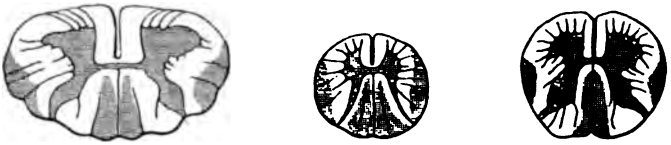
Under this name Gowers describes a group of cases in which there is a combination of posterior and lateral sclerosis. *Spastic ataxia* is the name preferred by Dana.

Ætiology.—The disease occurs in males more often than in females, and between the ages of thirty and forty, as a rule: but little is known as to its causation.

Symptoms.—It is slow in its onset, beginning in the lower extremities, which become weak, and show defective co-ordination at the same time. Thus, the patient readily tires, the muscles, especially the flexors of the hip and knee, yield when tested, and the gait is unsteady, with reeling when the patient turns or walks in the dark, and loss of balance when he tries to stand with the eyes shut. It differs from ordinary cases of locomotor ataxy in the absence of lightning pains, in the presence of ankle-clonus, and in the increase of the knee-jerk; the plantar reflex is more often either normal or increased. Sensation is mostly unimpaired; the pupil and optic disc remain normal; sexual power is often lost, and the sphincters may be slightly affected; the arms are more often free, but are sometimes involved in the same way as the legs. The disease is chronic in its course, with no very fatal tendency. As it progresses, the inco-ordination does not increase, but the weakness and rigidity become more marked, and the resemblance to spastic paraplegia becomes closer. Death results from intercurrent diseases, or occasionally from bladder complication or bed-sores.

The **Anatomical Change** in this disorder is simultaneous sclerosis of the lateral columns and of the posterior columns, but the extent and distribution of sclerosis vary considerably in different cases, and also differ slightly from what is common in locomotor ataxy and spastic paraplegia respectively. Thus, the posterior sclerosis affects the dorsal more than the lumbar region, and leaves the postero-external columns intact, or does not reach to the surface of the cord. In the lateral columns the sclerosis tends to advance to the anterior half, instead of remaining limited strictly to the area of the pyramidal tract. The cerebellar tract is also involved, and occasionally Burdach's or Türck's column, or the antero-lateral ascending tract.

FIG. 16.



Cervical. Dorsal. Lumbar.

Position of Lesion of Ataxic Paraplegia in Different Parts of the Spinal Cord.

Diagnosis.—This has to be made in the early stages from *locomotor ataxy*, especially by the excessive knee-jerk, the normal pupil, and the absence of lightning pains; and in the later stages from *spastic paraplegia*, by the presence or history of inco-ordination. It may also be confounded with *hereditary ataxy*, to which it is closely allied; and with disease (*e.g.*, tumour) of the *cerebellum*. In this last the muscular weakness is seldom so marked, and the local symptoms—headache and optic neuritis—should be sufficiently obvious.

Prognosis as to recovery is unfavourable.

Treatment must be similar to that of locomotor ataxy and primary spastic paraplegia.

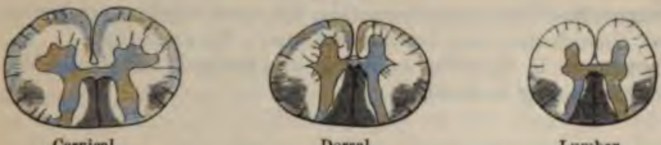
HEREDITARY ATAXY.

(*Friedreich's Ataxia.*)

The characteristic feature of this form of combined sclerosis is its congenital tendency and its appearance in several members of the same family. Thus, a man may transmit it to his children, or it may appear in two or more brothers and sisters without the parents being affected. It affects males only a little more often than females, though males may be especially affected in one family, females in another; and it is generally first noticed at an early age, either about the seventh or eighth year, or at puberty.

Symptoms.—The disease begins with inco-ordination in the lower extremities, which soon involves the trunk; and the inco-ordination is generally more jerky than in other forms, and presents a closer resemblance to the ataxy of cerebellar disease. The arms are affected later with a similar jerky irregularity, and the same extends to the muscles of the head and neck, so that there is some little resemblance to chorea. Muscular power is at first unaffected, but the knee-jerk is usually lost quite early, and the plantar reflex is also diminished. Sensation is often unaffected; sometimes there is slight anæsthesia. As a rule, lightning pains are not present. Speech is frequently impaired, producing hesitation; and syllables are dropped, and the movements of the tongue may be jerky. Nystagmus, or oscillation of the eyeballs, is often present; it is rather slow, lateral in direction, brought on by lateral movements, and checked when the eyes are fixed. In contrast with locomotor ataxy, this disease presents, as a rule, no ocular paralysis, no optic-nerve atrophy, no trophic changes, no visceral crises, and no affection of the sphincters. There is no mental change. As the disease progresses the muscles become weaker, especially those of the legs, and the flexors are more impaired than the extensors. Lateral curvature and club-foot may occur in consequence.

FIG. 17.



Cervical. Dorsal. Lumbar.

Sections of Spinal Cord from Case of Friedreich's Ataxia. (After *Newton Pitt.*)

The disease may last several years; death takes place generally from intercurrent affections, and not as a direct consequence of the spinal lesion.

Pathological Anatomy.—The essential lesion is a sclerosis of the posterior and lateral columns, but it differs somewhat from that found in ataxic paraplegia. The change is generally most intense in the lumbar region, and invades the whole of the posterior column. In the lateral column, the parts diseased are the pyramidal tract, the cerebellar tract, and the periphery of the

Myelopathic.—Disease of anterior cornua.

- Acute. Atrophic spinal paralysis, or poliomyelitis anterior acuta.
- Chronic. Progressive muscular atrophy, amyotrophic lateral sclerosis, and atrophies arising in connection with hypertrophic cervical meningitis, disseminated sclerosis, &c.

Neuropathic.—Disease of nerves.

- Neuritis, compression or other injury of nerves, and hypertrophic cervical meningitis.

Myopathic.—Disease of muscles.

- Pseudo-hypertrophic muscular paralysis and other forms of idiopathic muscular atrophy or dystrophy.

Our knowledge of the **Ætiology** of progressive muscular atrophy is very incomplete. The disease begins mostly in early adult life, between the ages of twenty-five and forty-five, and it is more common in males than in females. There appears to be a connection between muscular over-exertion and its commencement in some cases; and it is also attributed to mental distress and to exposure.

Symptoms.—In the large majority of cases (known as the Duchenne-Aran type) the disease begins in the upper extremities, and is seen first as a gradual atrophy of the short muscles of the thumb and little finger, so that the thenar and hypothenar eminences are flattened and disappear. The interossei muscles are also wasted, leading to depressions between the metacarpal bones; and when the atrophy is advanced, the shortening of the extensors, unresisted by the interossei, leads to the peculiar claw-like deformity (*main en griffe*) in which the first phalanges are over-extended on the metacarpals, and the middle and terminal phalanges are flexed on the first. Motor weakness accompanies, *pari passu*, the atrophy. These changes may occur in both hands, but often begin in one before the other, so that the hands are affected unequally. Other muscles are then involved; the deltoid is often the next to atrophy, but it may be the muscles of the upper arm, generally the biceps first, or of the forearm, when the extensors are affected before the flexors. The trapezius in its lower two-thirds, and the other scapular muscles, may be affected. The disease subsequently spreads to the trunk and neck, and the diaphragm and intercostals are sometimes involved, so as to cause serious difficulties in respiration. In the ordinary form of progressive muscular atrophy the legs are spared until very late; they may then be affected like the arms, or may be rigid, with or without wasting. The course of the disease is often exceedingly slow; it may be years before it spreads from the hands to the arms, and cord in front of this. The pyramidal fibres in the anterior column are sometimes affected, and Clarke's posterior vesicular column has been found degenerated in association with the cerebellar tract. The posterior nerve-roots are partially degenerated, but the peripheral nerves, as well as the anterior cornua of the cord, have hitherto been found free.

The explanation of the disease seems to be in a congenital want of vitality in the neuron-systems, or tracts involved.

The **Diagnosis** must depend upon the history of its association with other cases in the same family and its early appearance, the jerky unsteadiness of the head, the ataxy of the arms, the affection of articulation, and the nystagmus. By nearly all of these it is distinguished from ordinary *locomotor ataxy*. It may resemble, more or less, *ataxic paraplegia*, *disseminated sclerosis*, and *cerebellar disease*. In the first there is increased knee-jerk and no nystagmus; in the second the movements are more oscillating, less jerky, and the speech of a peculiar "staccato" character not present in Friedreich's disease; in the last there are accompanying head symptoms.

Prognosis.—This is unfavourable, the cases tending to become worse, though they may become stationary for some time.

Treatment is of little avail, but the methods suitable to locomotor ataxy may be tried.

PROGRESSIVE MUSCULAR ATROPHY.

This is a chronic disease characterised by wasting of muscles, with weakness consequent thereon, resulting from degeneration of the anterior cornua of the spinal cord. The names *wasting palsy* and *chronic anterior poliomyelitis* have also been used for it, but as it is not an inflammatory lesion, the latter is not suitable.

Before proceeding further it is desirable to make a few remarks on muscular atrophy in general. It has already been shown that the nutrition of the muscle depends on the integrity of its nerve-centre, the anterior cornu, and of the nerve-trunk proceeding thence to the muscle (lower motor neuron). In infantile paralysis there is a lesion of the *anterior cornua*, in neuritis a lesion of the *nerves*, and both are accompanied by muscular wasting. Further, there are cases of muscular wasting in which no lesion can be found in either cord or nerves, and which, so far as can be seen at present, result from a primary disease of the *muscles* themselves.

Thus we see that muscular atrophy, or *amyotrophy* (μῦς, muscle), may arise from disease of the spinal cord, of the nerves, or of the muscles, as shown in the following table:—

and nerve-trunks, and in the muscles themselves. The *anterior cornua* are scarcely, if at all, altered in size or shape, thus contrasting with the condition found in acute poliomyelitis; but they are pale, translucent, and almost entirely wanting in the proper nerve-elements, the large motor cells. Such of these as remain are smaller than normal, globular in shape, and without processes. At the same time the connective-tissue elements are increased. The posterior cornua are always normal. Sclerosis of the pyramidal tracts is present in many instances, and if it occurs early and is pronounced, the case may present all the features of amyotrophic lateral sclerosis (*see* p. 250). The anterior *nerve-roots* are visibly atrophied, being small and grey; but changes in the nerve-trunks are not so obvious, in consequence of the admixture of healthy fibres from the sensory roots. The *muscles* are pale and small. Under the microscope, the fibrillæ are found in different degrees degenerated. Some are simply diminished in size; in others the striation is indistinct, or replaced by fatty granules; in others there is marked longitudinal striation; and others again have undergone a vitreous or waxy change. The intervening connective tissue is increased in quantity.

Diagnosis.—Progressive muscular atrophy has to be distinguished from all other diseases accompanied by atrophy of muscles. The important feature is the slow commencement by atrophy and weakness together, without pain, spasm, or sensory troubles. This distinguishes it from *tumour* and *meningitis*, which may cause muscular atrophy. In *acute poliomyelitis* of children and adults the history is completely different. In the typical cases of *amyotrophic lateral sclerosis* the course is more rapid, and the reflexes are early increased. When the atrophy affects the hand alone, the deformity resembles somewhat the result of lesion of the *ulnar nerve;* but in this last the ulnar half of the hand is more decidedly affected (the radial lumbricales being supplied by the median nerve), and anæsthesia and trophic changes occur; in traumatic cases the history of injury will, of course, help. *Lead paralysis* is recognised by the extensors being first, and generally alone, affected; by the blue line on the gum, the detection of lead in the urine, and perhaps by the occupation, and preceding attacks of colic. *Multiple neuritis* is distinguished by the more rapid onset, the wide extent of the parts affected, the numbness or anæsthesia, and the tenderness of muscles. *Pseudo-hypertrophic paralysis* must be recognised by the enlargement of muscles, and its development in childhood; *idiopathic muscular atrophy*, by the atrophy beginning differently—*e.g.*, in the face or legs (*see* Diseases of the Muscles).

Prognosis.—This is unfavourable; but the progress of the atrophy is sometimes completely arrested.

again a period of rest may follow before other muscles are involved.

The electrical excitability of the muscles fails in proportion to the wasting. Usually faradic and galvanic excitability diminish together, but contractions can be still obtained, except when the wasting of any muscle is extreme. Then a partial form of reaction of degeneration may be found—*i.e.*, in the nerves, slightly diminished excitability to both currents; in the muscles, slightly diminished faradic excitability, and increased galvanic excitability, with slow contractions, and increase of ACC; or the reactions may fail altogether, that of the galvanic current lasting the longest.

The reflexes are commonly lost; the knee-jerk remains as long as the legs are unaffected. A characteristic and constant feature of this disease is the occurrence of *fibrillary contractions*, which were at one time thought to be pathognomonic; it is now known that they occur under other atrophic conditions. They consist of slight momentary twitchings of a few fibres of the muscle, visible on the surface, painless, though perceptible to the patient, and recurring every two or three minutes. They occur spontaneously, but may be brought out by a tap on the skin over the muscle.

Sensation remains intact, and the bladder and rectum are normal.

As already stated, the course is extremely slow, and death may take place from intercurrent diseases, such as phthisis, pneumonia, or bronchitis. But some cases are fatal through failure of the respiratory muscles, and in others the disease spreads upwards to the medulla oblongata, so that paralysis of the tongue, larynx, and pharynx results, constituting the *progressive bulbar paralysis*, which will be described shortly.

A *peroneal form* of progressive muscular atrophy has been described (Charcot and Marie, Tooth) which begins with atrophy of the peronei and anterior tibial muscles, and subsequently of other muscles of the foot and leg. It causes double talipes varus, and the limbs become cold and livid. Fibrillary tremors may or may not be present; reaction of degeneration is mostly found. It may extend to the muscles of the arm and hand, producing then the *main en griffe*. It occurs in members of the same family, and generally begins in early life.

Much doubt has attached to the pathology of the peroneal form; one of Charcot and Marie's cases recently examined *post mortem* showed atrophy of the anterior cornua, posterior sclerosis, and degeneration of the peripheral nerves.

Anatomical Changes.—In the ordinary form of progressive muscular atrophy (Duchenne-Aran) changes are found in the anterior cornua of the spinal cord, in the anterior nerve-roots and nerve-trunks, and in the muscles themselves. The *anterior cornua* are scarcely, if at all, altered in size or shape, thus contrasting with the condition found in acute poliomyelitis; but they are pale, translucent, and almost entirely wanting in the proper nerve-elements, the large motor cells. Such of these as remain are smaller than normal, globular in shape, and without processes. At the same time the connective-tissue elements are increased. The posterior cornua are always normal. Sclerosis of the pyramidal tracts is present in many instances, and if it occurs early and is pronounced, the case may present all the features of amyotrophic lateral sclerosis (*see* p. 250). The anterior *nerve-roots* are visibly atrophied, being small and grey; but changes in the nerve-trunks are not so obvious, in consequence of the admixture of healthy fibres from the sensory roots. The *muscles* are pale and small. Under the microscope, the fibrillæ are found in different degrees degenerated. Some are simply diminished in size; in others the striation is indistinct, or replaced by fatty granules; in others there is marked longitudinal striation; and others again have undergone a vitreous or waxy change. The intervening connective tissue is increased in quantity.

Diagnosis.—Progressive muscular atrophy has to be distinguished from all other diseases accompanied by atrophy of muscles. The important feature is the slow commencement by atrophy and weakness together, without pain, spasm, or sensory troubles. This distinguishes it from *tumour* and *meningitis*, which may cause muscular atrophy. In *acute poliomyelitis* of children and adults the history is completely different. In the typical cases of *amyotrophic lateral sclerosis* the course is more rapid, and the reflexes are early increased. When the atrophy affects the hand alone, the deformity resembles somewhat the result of lesion of the *ulnar nerve;* but in this last the ulnar half of the hand is more decidedly affected (the radial lumbricales being supplied by the median nerve), and anæsthesia and trophic changes occur; in traumatic cases the history of injury will, of course, help. *Lead paralysis* is recognised by the extensors being first, and generally alone, affected; by the blue line on the gum, the detection of lead in the urine, and perhaps by the occupation, and preceding attacks of colic. *Multiple neuritis* is distinguished by the more rapid onset, the wide extent of the parts affected, the numbness or anæsthesia, and the tenderness of muscles. *Pseudo-hypertrophic paralysis* must be recognised by the enlargement of muscles, and its development in childhood; *idiopathic muscular atrophy*, by the atrophy beginning differently—*e.g.*, in the face or legs (*see* Diseases of the Muscles).

Prognosis.—This is unfavourable; but the progress of the atrophy is sometimes completely arrested.

Treatment.—Drugs have but little value; arsenic and strychnia have seemed to do good sometimes. General hygienic treatment should be pursued: good air, nutritious food, exercise without strain, and freedom from mental worry. In addition to this the muscles may be locally treated with electricity, massage, and passive movements; but the improvement to be obtained by these means is at best but very slight.

AMYOTROPHIC LATERAL SCLEROSIS.

The lesion in this disease consists of degeneration of the motor cells of the anterior cornua, and sclerosis of the pyramidal tracts in the lateral columns—the combined lesions of progressive muscular atrophy and spastic paraplegia. The sclerosis of the pyramidal tracts extends through the medulla oblongata even to the crura cerebri; and the motor centres of the bulb (especially the hypoglossus and vago-accessory nuclei) are also affected towards the end of the disease in the majority of cases.

Ætiology.—It occurs between the ages of twenty-five and fifty, more frequently in females than in males (Charcot), but it cannot generally be referred to any particular cause.

Symptoms.—The disease begins with weakness in the upper extremities, which are soon seen to be affected with wasting. This is not limited to the interossei, or other muscles of the hand, though it may begin in them, but affects the whole upper extremity much more equally (according to Charcot) than in progressive muscular atrophy. Fibrillary contractions often occur, and the electrical reactions, as in progressive muscular atrophy, show only a simple diminution; unless the wasting is extreme, when reaction of degeneration may be present. Quite early in the history the tendon-jerks are increased, and can be elicited on striking the tendons of the biceps and triceps, or the lower ends of the radius and ulna. After a time rigidity takes place in the atrophied muscles, and considerable contractures may result. Charcot especially noted a deformity which he regards as characteristic of amyotrophic lateral sclerosis: the upper arm lies close along the body, the forearm is semiflexed and pronated, whilst the wrist is strongly flexed, and the fingers are bent into the palm. Generally, after eight to twelve months, the lower extremities become involved, presenting at first the characteristics of spastic paraplegia. Weakness and rigidity appear together, the former being masked by the latter. The knee-jerk is increased, and ankle-clonus can be obtained; the electrical excitability remains; and walking can be accomplished, though with difficulty. After some time, wasting also occurs in the lower extremities, but it is never so complete as in the upper. Sensation and the sphincters are unaffected. Finally, the disease spreads to the bulb, and atrophy of muscles in connection with nerve-centres quickly brings the patient into danger. The tongue, lips, and palate are atrophied and paralysed, deglutition and speech being rendered difficult; and implication of the vagus nucleus may affect the functions of respiration and circulation, so that an intercurrent bronchitis or pneumonia may carry off the patient. The duration is commonly from one to three years, and the prognosis is very unfavourable.

Diagnosis.—*Progressive muscular atrophy* has a slower course, with no excess of tendon reflexes or muscular rigidity. *Primary spastic paraplegia* begins generally in the lower extremities, and is unaccompanied by atrophy.

Treatment may be tried on the same lines as in these two diseases.

DISSEMINATED SCLEROSIS.

(*Multiple Sclerosis, Insular Sclerosis, Sclérose en Plaques Disseminées.*)

This disease is characterised by the development of numerous patches of chronic inflammation or sclerosis throughout the central nervous system. It has been described as occurring in three forms—cerebral, spinal, and cerebro-spinal; but the last is by far the most frequent. At an autopsy of such a case the surface of the spinal cord, medulla oblongata, pons Varolii, and base of the brain presents a number of irregular patches of pinkish-grey colour, rather sharply outlined, and contrasting with the natural white colour of the medulla, pons, and crura. On section, the discoloration is found to extend inwards so as to form deposits of a round or oval shape, ranging in size from that of a pea to that of a hazel-nut, generally harder than the normal nervous tissue, and even leathery, or cartilaginous; sometimes projecting above the level of the section, sometimes sunken below it. Recent patches are dark grey, older patches more yellowish-grey, and less translucent. They affect the white matter more than the grey matter; thus in the spinal cord the greater part of the cornua is unaffected, and in the cerebrum they are best seen on section of the hemispheres, which are dotted with the grey areas, and the walls of the lateral ventricles are often invaded. They are not frequent in the cerebellum; but the sclerosis may invade the olfactory bulbs, and the spinal and cranial nerve-roots. Under the microscope the outline of the patch or nodule is much less distinctly marked than it appears to the naked eye. The nodule consists chiefly of fibrous or finely fibrillated tissue, with some nuclei, granular corpuscles, and here and there still persistent axis-cylinders, although the majority of the nerve-fibres may be entirely destroyed. The walls of the vessels are also thickened, and surrounded with nuclei, or they may be in a state of fatty degeneration.

Ætiology.—The disease has no marked preference for one sex over the other. It occurs mostly in youth or middle age, and cases are recorded of the characteristic symptoms even in children. Cold, mental worry or excitement, injuries, and acute diseases have been assigned as causes.

Symptoms.—The onset of the disease is very variable; sometimes it is quite gradual, and the patients simply notice that they get weaker, or nervous, or tremulous, or spastic. In other cases there has been an apparently more rapid beginning—the knees have suddenly given way, or there has been sudden weakness of one arm or leg; this has perhaps recovered after a time, and then the same or another limb has become paretic. When the disease is fully developed the following three symptoms are nearly always prominent, and present a picture which is, on the whole, very characteristic. They are:—(1) *Tremor of muscles on attempting to move;* (2) a peculiar manner of speaking, *syllabic* or *scanning speech;* and (3) *nystagmus.*

The tremor is best observed in the hand and arm when the patient attempts to take hold of an object—the limb oscillates irregularly to the right and left, or up and down, with regard to the object aimed at, the excursions from the straight line being often several inches in extent. When the patient sits up in bed, or stands up, the body swings to and fro, and the head undergoes a series of nodding movements; while in the attempt to walk the movements of the legs are similarly unsteady. When the patient is lying quiet in bed, or sitting with the back, head, and arms supported, he is perfectly quiet. The movements are more violent the greater the effort, and the more the patient feels that he is under the observation of others. The limitation of the tremors to the time of attempted movements has given them the name of *intention tremors.*

In talking, every syllable is distinctly uttered in a slow, deliberate manner and somewhat suddenly, as in the *staccato* delivery of music. There is little or none of the natural slurring of some syllables and accentuation of others. The voice is also rather high-pitched and monotonous. It has been observed with the laryngoscope that the vocal cords are apt to relax in phonation, and to undergo rapid changes of tension.

The *nystagmus*, or oscillation of the eyeballs, is, like the movement in the limbs, only brought on by voluntary movements—that is, when the eyes are fixed upon an object, or when they are much turned to one side. It is thus unlike the oscillation seen in some cerebellar tumours, or the constant movement which occurs in some diseases of the fundus of the eye.

The motor power is generally diminished, and the knee-jerks are exaggerated; and sooner or later rigidity of the lower extremities occurs, when ankle-clonus will also be observed. The rigidity is pronounced in some advanced cases, where it may co-exist with actual paralysis, from the sclerosis invading nearly the whole thickness of the cord at one spot, and thus, like a transverse myelitis, producing paraplegia. But in earlier stages rigidity with extension and adduction of the legs may be a troublesome symptom, and the gait will have a *spastic* character. Occasionally, atrophy of muscles is observed, when it may be supposed that sclerosis has invaded the grey cornua. Mostly electric irritability is normal; only in later stages it may be diminished, or the reaction of degeneration may be observed, where muscular atrophy is present. There may be modifications of sensation, such as numbness or formication, but rarely complete anæsthesia.

The bladder, rectum, and sexual organs often retain their power, or there may be some impairment of the functions of the bladder—involuntary expulsion or slight retention. There is sometimes defect of vision, with diminution of the field and achromatopia, and rarely complete blindness. Primary optic atrophy is not uncommon. Headache and vertigo are occasionally present. More frequent is some impairment of the mental or emotional powers. The patients are especially liable to laugh or cry without apparent reason, and the intelligence is weakened as the disease progresses. Delusions of grandeur may occur, and the case may ultimately assume all the characteristics of general paralysis of the insane. In some instances peculiar apoplectic attacks occur. The patient is seized with headache, giddiness, then loss of consciousness, and weakness of the arm and leg on one side. The face is red, the pulse is frequent, and the temperature rises to 104° or 106°. Recovery takes place in a day or two. Epileptiform attacks are more rare. Occasionally, the usual symptoms may be absent, and death takes place from an apoplectic attack; and in others, from special localisation of the patches, the various chronic spinal diseases, myelitis, spastic paraplegia, or bulbar paralysis may be simulated. But it is remarkable how constantly the three symptoms first mentioned are the prominent features of the case.

The disease may last several years—ten, fifteen, or twenty. Death may take place much earlier from an apoplectic seizure, or the patient may be bed-ridden for years with paraplegia, and die from the accidents associated with that condition or from intercurrent disease.

Pathology.—The symptoms are not at present satisfactorily explained. The tremors have been referred by some to the want of "insulation" of the axis-cylinders in the sclerosed patches, so that impulses are irregularly transmitted. Others attribute the symptoms to the patches which invade the pons Varolii or the higher parts of the motor tracts.

Diagnosis.—This is easy when the three cardinal symptoms are alone prominent. The tremors can scarcely be confounded with chorea or paralysis agitans. In *chorea* the movements are more twisting, jerking, or writhing, and occur during rest. In *paralysis agitans* they are regular and rhythmical, more rapid, and less extensive; they occur during rest, and may, in early stages, be stopped by voluntary effort. The oscillating movements which are seen in disease of the *cerebellum* closely resemble those of sclerosis, but the former may be distinguished by the other evidences of local disease. It may be, as already implied, that they have the same origin. Charcot says that *chronic cervical meningitis* with cortical sclerosis may produce similar tremors. *Mercurial tremors* resemble disseminated sclerosis in the movements of the limbs, but the head and trunk are less affected in the former. In particular cases there may be some difficulty in distinguishing the disease from the chronic spinal complaints already mentioned, especially if, as is sometimes the case, the tremor is absent (*see also* Acute and Chronic Myelitis, pp. 222, 224). In the early stages patients may be thought to be hysterical, or, on the other hand, *hysteria* may simulate sclerosis.

The **Prognosis** is bad, and the **Treatment** mainly symptomatic. Any direct treatment would naturally be similar to that of locomotor ataxy or chronic myelitis.

SPINAL MENINGITIS.

The spinal membranes may be affected separately; but it is common for inflammation beginning in one to spread to the other. Inflammation of the pia mater is called *leptomeningitis*, that of the dura mater *pachymeningitis*. The forms of inflammation commonly recognised are—first, an inflammation of the outer surface of the dura mater, usually set up by disease in the neighbourhood—this is called *meningitis externa*, or *pachymeningitis externa;* secondly, inflammation of the internal surface of the dura mater, generally chronic, and resulting in great thickening of the membranes (*pachymeningitis interna hypertrophica*), or in extravasation of blood, and the formation of fibrinous, pigmented deposits (*pachymeningitis interna hæmorrhagica*); lastly, inflammation, beginning in the pia mater, or *leptomeningitis*, which may be acute or chronic. It will be seen that the symptoms of all these forms present much similarity, as they depend not so much on any alteration in the membrane itself, as upon the implication of the nerve-roots which pass through them, and of the cord which they enclose.

ACUTE LEPTOMENINGITIS—ACUTE SPINAL MENINGITIS.

Ætiology.—Its causes are often obscure, but amongst those to which it can be traced are exposure to cold, sunstroke, injuries such as fractures and dislocation of the spine, and the puncture of a spina bifida. It has sometimes arisen in connection with pneumonia, or one of the infectious diseases, as scarlatina, typhoid, or septicæmia, or the puerperal state. Inflammation may spread from outside the spinal canal, first causing an external meningitis, or from the cerebral to the spinal membranes. Further, a tubercular spinal meningitis not infrequently accompanies tubercular meningitis of the brain; and the two membranes are associated together in the disease known as epidemic cerebro-spinal meningitis or cerebro-spinal fever (p. 82).

Symptoms.—An attack of acute spinal meningitis usually begins with rigor and elevation of temperature; there is severe pain in the back, which may be localised, or extend along the whole length, and this pain is usually increased by movement as well as by pressure. In addition, there are pains of shooting, darting, paroxysmal character, radiating in the course of the nerves arising from the part; and hyperæsthesia of the skin, and even of the muscles, in the same areas. Irritation of the anterior nerve-roots leads to spasm of the muscles, producing rigidity of the spine, with more or less severe arching, or *opisthotonus*. This may be present in the whole length of the spine, or affect the neck alone, when the occiput is fixed back between the shoulders (retraction of the head, cervical opisthotonus). The abdominal muscles and the muscles of the legs are also affected by spasm, and the spasms are increased by attempts at movement. The reflexes are generally increased. The bowels are constipated, and the urine is retained by spasm of the sphincter, or later by paralysis of the detrusor. There are the usual accompaniments of pyrexia; the temperature is generally raised, the pulse is quick, and the "tache méningitique" (p. 299) is well marked. After some time—it may be a few days or longer—the symptoms of irritation, pain, and spasm give place to those of paralysis, anæsthesia, and diminished reflexes; and the case then approximates to one of myelitis, and is either rapidly fatal from exhaustion, paralysis of the respiratory muscles, or acute bed-sores; or lapses into a more chronic condition, with atrophy and shortening of muscles. Some patients recover after several months; others die from exhaustion, bed-sores, or vesical and renal complications.

Spasm of
muscles

The symptoms of meningitis vary considerably, and are not always so manifest as above indicated. The signs of irritation may be of very short duration, and those of paralysis become prominent quite early; and in cases where the cerebral membranes are involved at the same time, the spinal symptoms may be entirely masked, as is frequently the case in tubercular meningitis. In cerebro-spinal fever the symptoms of the two conditions are combined. Further, differences are observable according as the inflammation affects mainly the cervical, the dorsal, or the lumbar region. It is sufficient here to point out that, in cervical meningitis, retraction of the head, dyspnœa from implication of the diaphragm, dysphagia, inequality of the pupils, and great elevation of temperature are likely to occur; in dorsal meningitis the trunk muscles, and in lumbar meningitis the muscles of the loins and legs, are especially affected. The duration varies from two or three days to three weeks in the more acute cases, and is occasionally much longer.

Morbid Anatomy.—In the early stage the surface of the pia mater is reddened from increased vascularity, and small hæmorrhages may be present; an exudation then takes place in the meshes of the pia mater and upon the surface, at first greyish in colour, subsequently purulent and yellow or greenish-yellow. This deposit is seen often over a large extent of the cord in irregular patches, more upon the posterior than the anterior aspect from the effects of gravitation. The spinal fluid is turbid and opaque. The inflammation affects also the inner surface of the dura mater, and the two membranes may be united by the viscid purulent lymph. The process frequently extends into the cord, affecting especially its periphery, in which dilated vessels and leucocytes occur, and the nerve-roots are similarly involved.

Diagnosis.—Meningitis has mainly to be distinguished from myelitis and from tetanus. As contrasted with meningitis, there is less fever in *myelitis*, the local and radiating pain, rigidity, and spasms are less marked or absent, and paralysis and anæsthesia come on quite early. *Tetanus* is distinguished by the persistent spasm, aggravated on the slightest peripheral irritation, by its commencement in trismus, by only slight pyrexia, if any, and by the history of wound in the majority of cases. Rheumatism of the lumbar muscles or of the vertebral articulations may to a certain extent simulate meningitis.

Treatment.—In the acute stage the treatment is similar to that of acute myelitis, with this addition—that perfect rest and avoidance of all external irritations should be ensured; while morphia, chloral, and potassium bromide, or chloroform inhalations, should be given to relieve pain. Mercury and potassium iodide may be used internally, or the oleate of mercury may be rubbed into the spine. In the less acute stages, mercurials and potassium iodide are to be given internally and counter-irritants may be applied to the spine.

Fibrillary

Chronic Leptomeningitis.

Ætiology.—The chronic form of inflammation may be only the continuation of an acute process; or it may be chronic from the beginning, and has in that case been attributed to cold, syphilis, chronic alcoholism, and sometimes strains or injuries. It is commonly found in association with chronic inflammatory (or degenerative) processes in the cord itself, such as chronic myelitis and locomotor ataxy; and it may also result from lesions external to the cord.

Anatomy.—The condition is one of fibrous thickening of the pia mater, with dilatation and thickening of the walls of the blood-vessels. Its distribution may be extensive when it follows upon an acute lesion, more scattered when it is chronic from the first. The parts affected are in some cases determined by the pre-existing lesion of the cord. The pia mater and arachnoid may be adherent to the dura mater: the cord itself often shows inflammatory changes in the connective tissue at the surface (annular myelitis), or a more extensive myelitis; and the nerve-roots may be compressed and atrophied.

Symptoms.—These are the same in kind as those of acute meningitis—namely, symptoms due to local irritation of the membranes, others due to implication of the nerve-roots, and others again which result from implication of the cord; but they are gradual in their onset, they are not accompanied by fever, the muscular spasms and rigidity are much less marked, than in the acute form, the sensory symptoms are often more localised, and paralysis may occur early. Ultimately the case may assume all the features of chronic myelitis.

Internal Pachymeningitis.

It has been already stated that the inner surface of the dura mater is often involved in both acute and chronic leptomeningitis; but there are two forms of pachymeningitis which require separate mention—namely, *pachymeningitis interna hypertrophica* and *pachymeningitis interna hæmorrhagica*. In the former the dura mater becomes immensely thickened, so as to cause very severe compression of the cord and nerve-roots. Charcot has described it as especially affecting the cervical region (hypertrophic cervical pachymeningitis). After a first stage of shooting pains in the back of the neck, shoulder, arms, and upper part of the thorax, with muscular twitchings and spasms, there are gradually developed anæsthesia, paralysis, and atrophy, and loss of electrical reactions in the distribution of the compressed nerve-roots. Charcot observed that the median and ulnar nerves were more affected than the musculo-spiral, and that consequently there

Fibrillary contractions

arose a deformity of the upper limb, characterised by extension at the wrist and metacarpo-phalangeal joints, and by flexion of the phalanges. This appears to be due to implication of the lower part of the cervical region. In a case affecting the upper part of the cervical region the muscles supplied by the musculo-spiral were paralysed, and a condition of extreme flexion was the result. As the compression of the cord increases, paraplegia, secondary degenerations, and spastic rigidity of the paralysed parts occur. In rare cases, hypertrophic pachymeningitis affects the lumbar region, or the cauda equina, when the pains, paralysis, and atrophy are situate in the lower extremities, and the sphincters are involved early.

In *pachymeningitis interna hæmorrhagica*, or *hæmatoma of the dura mater*, the inner surface of the membrane is covered with a reddish-brown exudation, consisting of fibrin, connective tissue, recent extravasations of blood, and, it may be, cysts containing blood in different stages of change. This form of meningitis affects the greater part of the cord, and is associated mostly with a similar condition in the cerebral dura mater (*see* p. 304). The disease has been attributed to the abuse of alcohol, and it is seen in some cases of mental disorder.

The **Symptoms** are those of a slight chronic meningitis, but they are often masked by others which result from the cerebral lesion.

EXTERNAL PACHYMENINGITIS.

As already stated, this is mostly secondary, and arises from any inflammation in the neighbourhood of the dura mater: thus, deep bed-sores over the sacrum may slough into the spinal canal, or the dura mater may be inflamed by caries of the spine (the most common cause), by retro-pharyngeal abscess, or abscess in other situations, by cellulitis of the neck, or inflammation of the sub-pleural tissue; or it may result more directly from injury. It may be acute or chronic. In the acute form, such as occurs from sloughing bed-sores, the external surface of the dura mater is reddened, or presents lymph upon the surface, or may be covered with a layer of pus. In caries of the spine the process is generally more chronic, and the outer surface of the membrane is covered with caseous or semi-caseous deposit, and the membrane is itself thickened. More or less inflammation or compression of the nerve-roots commonly co-exists, and the cord may be much narrowed. In the purulent cases especially the process may extend to the internal surface.

Symptoms.—These are not essentially different from those already described under Leptomeningitis. They are pain in the back at the seat of the lesion, stiffness of the back from rigidity of the muscles, and pain on movement, pains in parts corresponding to the nerves arising from the region affected, hyperæsthesia, jerking and tension of the muscles, and later, the symptoms of compression of the cord—namely, paralysis and anæsthesia, in varying degrees, of the parts below the seat of the lesion. In acute cases the symptoms of irritation are likely to predominate; in chronic cases those of compression.

Diagnosis.—The important point is the recognition of an external cause, otherwise it may be impossible to distinguish it from other forms of spinal meningitis.

The **Prognosis** is unfavourable in acute cases, but more hopeful in caries of the spine, of which a large number of cases make a more or less perfect recovery.

Treatment.—The chief indication is to remove the original cause, if possible; the others, to aid in the absorption of inflammatory products, and to treat the myelitis resulting from compression.

SPINAL MENINGEAL HÆMORRHAGE.

This is a rare occurrence. It arises from injuries, such as blows, stabs, falls on the feet or back, and traction on the spine of newborn children during delivery. Blood effused into the cranial cavity sometimes runs down into the spinal canal; and, very rarely, aneurysms of the aorta or of the vertebral artery have burst into it. Hæmorrhage into the membranes may form part of purpura and scurvy, or of other general conditions characterised by a tendency to hæmorrhage; and, lastly, the pachymeningitis hæmorrhagica, just described, is associated with hæmorrhage. Like hæmorrhage into the cord itself, its symptoms are distinguished by the suddenness of their onset. There are severe pain in the back, pains radiating along the nerves, and spasm and rigidity of the muscles supplied by the nerves. Subsequently there is loss of power and sensation, and in some cases complete paralysis and anæsthesia occur. The paralytic symptoms follow quickly upon the signs of irritation, and reach their height in a period varying from a few hours to a few days. Death, also, may occur in a few hours, or symptoms characteristic of meningitis may supervene.

Diagnosis.—Spinal meningeal hæmorrhage is distinguished from *hæmorrhage into the cord* chiefly by the fact that signs of irritation, such as muscular cramps, spasm, rigidity and hyperæsthesia, precede paralysis, and the paralysis is less complete. *Spinal meningitis* is more gradual in its onset, and is accompanied by fever from the first.

Prognosis.—This is sufficiently serious, and many cases are fatal. On the other hand, recovery is more frequent than in hæmorrhage into the cord.

Treatment.—This must be conducted in the same way as that of intra-medullary hæmorrhage. The later treatment is that of spinal meningitis.

TUMOURS OF THE SPINAL CORD AND ITS MEMBRANES.

Tumours may grow in the spinal cord, in the spinal membranes, or from parts of the spinal canal outside the membranes. Of these latter (*extra-dural*) growths, the most common are tumours growing from the vertebræ, such as sarcoma and carcinoma: but the inflammatory material resulting from caries of the spine also forms masses which act like tumours in compressing the cord. Fatty tumours and hydatid cysts occur outside the membranes, but are rare. Within the membranes (*intra-dural*) the following growths occur: syphiloma, sarcoma, myxoma, lipoma, myolipoma, fibroma, tubercular tumours, hydatid cysts, and cysticercus. The first three are the most common. Some of these tumours grow from the *nerve-roots*, especially sarcoma, myxoma, fibroma, and glioma. The tumours arising within the *cord* itself are glioma, syphiloma, sarcoma, myxoma, and tubercular masses (*see* Tumours of the Brain).

The tumours are generally isolated and of small size. There may be two or more tubercle-masses, syphilitic gummata are occasionally multiple, and there may also be several neuromata at the same time. Tumours of the cauda equina may attain a considerable size from the greater space allowed them. A glioma forming around the central canal (peri-ependymal glioma) and extending a variable distance along the cord may by its softening lead to the formation of an elongated cavity or *syringomyelia* (*see* p. 262).

In their continued growth the tumours produce important effects upon the cord and nerves. Extra-medullary tumours cause compression and wasting of the nerve-roots, or press upon the cord and set up myelitis and subsequent secondary degenerations. Similarly, myelitis and secondary degenerations may follow the growth of tumours in the cord itself. Occasionally the vessels of a glioma rupture, and the symptoms of a spinal hæmorrhage develop with their usual suddenness.

Ætiology.—The causation of spinal tumours cannot be treated of apart from that of tumours in general. It is doubtful whether some cases have been due to blows or injuries. Tubercle and gumma occur as parts of a general morbid condition. Some tumours are probably congenital (*e.g.*, myolipoma).

Symptoms.—The symptoms of a spinal tumour vary with its situation.

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ırs outside the cord give rise to symptoms indicating irrita- ompression of the nerve-roots, especially local pain in the .ins shooting from the same spot in the direction of spinal ·igidity of spinal muscles, hyperæsthesia or localised anæs- nd motor weakness. These symptoms may be unilateral, ted to one or other region of the spinal cord—cervical, r lumbar. After some time—it may be months or a year -symptoms of compression of the cord or local myelitis :onsisting of paralysis and anæsthesia below the seat of ›n, with spastic rigidity, increased reflexes, and vesical In multiple tumours the symptoms may be more varied; ›e already mentioned (p. 239) tabes dorsalis was clearly d. Ultimately, in nearly all cases the termination is fatal, ame way as in myelitis—either through failure of the ›ry muscles, or through bed-sores and exhaustion or pyæmia, gh cystitis and renal complications.

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Prognosis is very unfavourable. Gummata may yield again and again, or entirely, to treatment, and tubercles may possibly in a few cases become obsolete; but in the majority of instances the disease can only terminate fatally in the manner above indicated.

Treatment.—Where there are good grounds for a diagnosis of gumma, vigorous anti-syphilitic treatment should be employed; that is, full doses of potassium iodide, and mercurials—either the perchloride internally, or the ointment or oleate rubbed into the skin. Even when the syphilitic nature is uncertain the same treatment may be for a time tried, since other forms of tumour are not amenable to any drugs, with the exception of tubercle-masses, in which case the small chance of recovery may be improved by general tonics, iron, cod-liver oil, and similar remedies. The cases will generally require symptomatic treatment, like other instances of paraplegia; bed-sores and vesical lesions must be guarded against, pain alleviated, &c. Surgical assistance may be considered in the case of tumours, which are not actually within the cord itself, and a few have been successfully removed, since Horsley's first case in 1890.

SYRINGOMYELIA.

The elongated cavities occasionally found in the spinal cord may be a simple slight dilatation of the central canal of the cord (*hydromyelia*), or a more definitely pathological condition called syringomyelia (σῦριγξ, a pipe). In the latter there is usually a single cavity, occupying the cervical and upper dorsal regions, lying in the posterior half of the cord behind the commissure, or in one or other posterior cornu. It is elongated, several inches in vertical extent, and is very variable in diameter at different levels of the cord. On transverse section it may have the appearance of a fissure running transversely from side to side, or may form a circular or oval space, occupying the greater part of the posterior half of the cord. At one or more points in its length it may actually be divided into two, and sometimes a section will show that each posterior cornu presents a cavity; but with these exceptions it is mostly one long tube, suggesting that it has arisen from a dilatation of the central canal; and this seems to be supported by the presence in some parts of an epithelium on the inner surface, and the occasional advance of the cavity sufficiently forwards to include the central canal. But the central canal is often present in its normal condition, and hence this explanation requires modification. The cavity is often bounded by a thin layer of dense fibroid

Within the cord, at first gradually displacing parts, it will at length produce sufficient compression to interrupt conduction, and paralysis and anæsthesia below the lesion will slowly develop. The paralysis often precedes the loss of sensation. If the tumour is limited to one side, the symptoms will be unilateral, with paralysis of one side, and anæsthesia of the other. If it involves grey matter to any extent, there will be atrophy of the nerves and muscles proceeding from the part. Ultimately, the tumour, by continued growth only, or by setting up myelitis, is likely to cause complete paralysis, with secondary degenerations, and increased reflexes and rigidity of the muscles of the lower extremities. If the tumour is situate in the lumbar enlargement of the cord, the muscles are atrophied, the reflexes are lost, the electrical reactions are altered, and the sphincters are implicated.

Tumours outside the cord give rise to symptoms indicating irritation or compression of the nerve-roots, especially local pain in the spine, pains shooting from the same spot in the direction of spinal nerves, rigidity of spinal muscles, hyperæsthesia or localised anæsthesia, and motor weakness. These symptoms may be unilateral, and limited to one or other region of the spinal cord—cervical, dorsal, or lumbar. After some time—it may be months or a year or two—symptoms of compression of the cord or local myelitis appear, consisting of paralysis and anæsthesia below the seat of the lesion, with spastic rigidity, increased reflexes, and vesical troubles. In multiple tumours the symptoms may be more varied; in a case already mentioned (p. 239) tabes dorsalis was clearly simulated. Ultimately, in nearly all cases the termination is fatal, in the same way as in myelitis—either through failure of the respiratory muscles, or through bed-sores and exhaustion or pyæmia, or through cystitis and renal complications.

Diagnosis.—This can, of course, be only made after a certain time has elapsed. The distinctive features are pain and other symptoms of irritation, limited to one or two segments of the cord, followed by progressive paralysis, without any evidence of caries or injury to the spine. Tumours in the cord itself may cause paralysis, without preceding pain or spasm, and the symptoms are early bilateral; if the tumour grows in the membranes, the symptoms may be limited to one side for a long time. The diagnosis from *myelitis* depends chiefly on the slow development of the symptoms and their strict limitation to one spot. The nature of the tumour may be difficult to determine: a syphilitic history and the association of cerebral symptoms indicate gumma; and preceding cancer may give a clue in some cases. Tubercle occurs in early life, and may be suggested by a strong family history or by indications in the patient; but a tubercle of the cord is infinitely rare as compared with the frequency of a phthisical tissue, and surrounded by tissue which is translucent, gelatinous, deficient in nerve-fibres or other structural elements, and consists of neuroglial tissue of an embryonal character. In some cases there is a growth of sarcoma or glioma in the tissue around the cavity.

The probability seems to be that the neuroglial tissue, and sometimes the cavities, are congenital; the cavities, at any rate, have been found in young children in association with hydrocephalus, or distension of the cerebral ventricles, as in a case recorded by me. They may then be due to the inadequate filling-up of the posterior portion of the cavity which is formed from the primitive groove: and around this cavity the embryonal tissue persists. In later life there is both an increase of neuroglial or gliomatous tissue, and an enlargement of the cavity.

Symptoms.—The condition is not always accompanied by symptoms, and it has often been discovered *post mortem* when not suspected. In other cases there has been a great variety of symptoms, which have come on insidiously, and generally first in early adult life. The most constant results are as follows. There is loss of sensation to pain, heat, and cold, while sensation to touch, the muscular sense, and the special senses is unaltered; as a result of this patients may suffer burns and injuries which they would escape under normal conditions. Muscular atrophy, progressive in its development, affects the upper extremities especially. The small muscles of the hands are wasted, producing the *main en griffe*, and subsequently the forearms and higher parts are involved, giving an appearance like that of progressive muscular atrophy (*see* p. 247); the legs are less often implicated, but they may be weak or become slightly spastic. A third feature of interest is the occurrence of trophic disturbances, such as œdema of the fingers, bullæ, ulcers, whitlows (*Morvan's disease*), loss of the nails, and brittleness of the bones. The joints, especially the shoulder and elbow, may present changes very like those described under locomotor ataxy; the ligaments and capsule become relaxed, and the head of the bone is absorbed. Spinal curvature, and contraction of the palpebral fissure, with retraction of the eyeball and small pupil also occur. The bladder and rectum escape, as a rule. Rarely, the lesions spread to the medulla and pons so as to produce paralysis of some of the muscles supplied by the cranial nerves. The cases often last for years, and die from secondary complications.

Diagnosis.—The conditions most likely to be confounded with these more typical cases are multiple neuritis and progressive muscular atrophy. In the former the legs are more decidedly paralysed; in the latter there are no sensory troubles.

Treatment.—This can be little else than symptomatic.

COMPRESSION OF THE SPINAL CORD.

A few words may be said of compression of the cord in general, although it has been frequently alluded to in the descriptions of myelitis, meningitis, and tumours. The most common cause of compression of the cord is undoubtedly caries of the spine, not from "angular curvature," which the caries produces, but from the inflammatory or caseous products, which form between the diseased bone and the external surface of the dura mater, destroying the posterior common ligament and setting up an external pachymeningitis. The other less frequent causes are tumours growing from the bones or membranes, aneurysm eroding the spinal column, and chronic thickening of the membranes (hypertrophic meningitis).

Whatever the cause of the compression is, the cord is narrowed, it may be to one-half or one-third its diameter, and myelitis is set up in the compressed part and in the cord immediately adjacent. Subsequently, degeneration takes place in the posterior columns above the lesion, and in the pyramidal tracts below the lesion. To the naked eye and the microscope the changes are like those which are seen in myelitis from other causes—destruction of nerve-elements and increase of connective-tissue; but even after prolonged and considerable compression it is remarkable that many nerve-fibres may remain intact.

Symptoms.—These are determined partly by pressure upon the nerve-roots, and partly by pressure upon the cord itself. The former occur first, and consist of pains of burning or neuralgic character in the area of the nerves whose roots are compressed. They differ from true neuralgia, according to Charcot, in not possessing *points douloureux*. Accompanying them is hyperæsthesia, at points corresponding to the distribution of the nerves; and later on, anæsthesia supervenes, and may coexist with the severe pains, constituting the so-called *anæsthesia dolorosa*. Occasionally, trophic disorders of the skin are present, such as zona, bullæ, or eschars. Compression of the anterior nerve-roots may lead to atrophy of muscles, weakness, and sometimes, but not often, contracture. In cancer of the spine this compression of the nerve-roots is often brought about in a peculiar way; the laminæ of the vertebra, softened by the cancer-growth, give way under the weight above them, and fall in upon the nerves lying in the intervertebral foramina. The symptoms due to direct compression of the cord are those with which we are familiar in transverse lesions: paralysis, anæsthesia or other modification of sensation, increased reflexes, often some vesical trouble, and generally spastic rigidity of the paralysed muscles. The relation of anæsthesia to paralysis varies much in different cases, and in the same case at different times. Loss of motion is, as a rule, the most prominent symptom, and anæsthesia may be entirely absent. The activity of the reflexes is often in excess of the motor paralysis. It is another important feature, when the compression results from caries, that recovery may take place completely, or improvement may again be followed by relapse. The site of the compression, of course, determines some differences in the symptoms. Compression *limited to one side* will cause the pains to be unilateral, and the paralysis may be on the same side, the anæsthesia on the opposite, as has been stated to be the result of strictly one-sided lesions (*see* p. 213). *Cervical* compression may be accompanied by alterations of the pupil, especially dilatation from irritation of the cilio-spinal centre, by cough and dyspnœa, dysphagia, vomiting, or very slow pulse. The distribution of the paralysis is also sometimes striking; all four limbs may be paralysed, the upper limbs being wasted, with diminished reflexes, as a result of compression of the lower motor neurons. But the arms may be paralysed as a result of compression of pyramidal fibres, and the muscles will then preserve their volume and their electrical reactions, while the reflexes are increased. In some such cases the legs remain unaffected. The distinctive features of compression of the *lumbar* region are paralysis, with flaccidity, wasting, diminution of the reflexes, paralysis of the sphincters, and tendency to bed-sores.

The **Diagnosis** of compression-paraplegia depends largely on the pain indicative of nerve-compression in association with the weakness indicating pressure on the cord. Often the spine is tender at the seat of disease, and the back should always be examined for the prominence which caries so often produces (*kyphosis*, angular curvature). It has been already stated that the compression is not generally dependent upon the curvature, but on the inflammatory products about the diseased bone; and, as a fact, all the symptoms of compression may precede by several months the appearance of any projection of the spine.

Prognosis.—Even without operation, improvement may occur in cases of caries, and recovery takes place sometimes after months or years.

Treatment.—This consists in the removal of the cause. The treatment of tumour has been mentioned; cancerous growths are beyond our resources at present. Paralysis from caries requires prolonged rest, with sea air, nutritious diet, cod-liver oil, and tonics; and when improvement is well established, the spine should be supported by a plaster-of-Paris or felt jacket for some time afterwards. In some cases, however, the pus and caseous inflammatory products have been successfully removed by the operation of laminectomy.

DISEASES OF THE MEDULLA OBLONGATA.

The medulla oblongata is subject to similar diseases with other parts of the central nervous system, such as hæmorrhage, inflammation (bulbar myelitis), and tumours. The symptoms are determined by the anatomical structure of the medulla oblongata, which, besides transmitting the motor and sensory tracts, contains the special nerve-centres of the lower cranial nerves, from the fifth to the twelfth. Hence, on the one hand, there may be paralysis of the trunk and limbs; on the other, impairment of the functions of phonation, articulation, mastication, and deglutition. These symptoms make up what is commonly called "bulbar paralysis"; and it will be best to describe first a chronic form known as progressive bulbar paralysis, and subsequently the more acute results of hæmorrhage and embolism, and the effects of the growth of tumours.

PROGRESSIVE BULBAR PARALYSIS.

(*Labio-glosso-laryngeal Paralysis.*)

In this disease there is a slowly developed paralysis of the lips, tongue, larynx, and pharynx, resulting from degeneration of the nuclei of the nerves which supply the muscles of these parts.

Ætiology.—It occurs in middle and advanced life, between the ages of thirty and seventy, and is more frequent in men than in women. It is not always possible to attribute it to any cause; but exposure to cold, falls or other injuries involving the neck, and syphilis have occasionally been credited with its production. Bulbar paralysis is intimately related to progressive muscular atrophy and amyotrophic lateral sclerosis, occurring often as the last stage of either of those diseases; and sometimes an illness which has begun as bulbar paralysis may later affect the spinal centres.

Symptoms.—The disease is generally first evident in the movements of the *tongue*, and the articulation of sounds which depends upon it becomes faulty. These are, first of all *e*, and then *s*, *l*, *k*, *g*, *t*, *d*, *n*, *r*, and *sh*. The paralysis increases, and may become so complete that the tongue cannot be protruded, and lies at the bottom of the mouth. After a time atrophy takes place, and the organ becomes wrinkled and furrowed. Fibrillary contractions are often observed in it. Shortly after the tongue begins to be

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paralysed, the same change occurs in the *lips*. The articulation of *o*, *u*, *p*, *f*, *b*, *m*, and *v* is impaired, and whistling, blowing, and pouting are performed with difficulty. The lower lip drops away from the teeth, the naso-labial folds are more marked, and saliva dribbles from the angles of the mouth. Atrophy here also can be detected in the lips becoming thinner, and fibrillary contractions may be seen. Food also collects between the teeth and the cheek; but the paralysis of the facial nerve is confined to the lower half of the face, the upper half remaining entirely free. After the tongue and lips, the *palate* is paralysed, and, as a result, liquids may regurgitate through the nose, and the voice acquires a nasal quality. The articulation of *o* and *p* is also impaired by this, since the volume of air which is required for their production is diminished by its escape into the nasal cavity. Paralysis of the *larynx* produces hoarseness and, finally, complete aphonia; and during swallowing food is apt to enter the larynx from the paralysis of the tongue and certain muscles of the larynx (arytænoideus, thyro-ary-epiglottideus, and thyro-arytænoideus externus), by the combined action of which the two passages should be cut off from one another. As a result, choking takes place, and small particles are inhaled, which may set up bronchitis or lobular pneumonia. The laryngeal paralysis also renders coughing and hawking difficult or impossible. Dysphagia is further aggravated by paralysis of the *pharyngeal* muscles.

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The progress of the disease is very slow, but the condition of the patient in an advanced stage is highly characteristic. The lower lip falls; from the angles of the mouth dribbles saliva, which the patient is constantly wiping away with a pocket-handkerchief; the tongue cannot be protruded, and the only sound uttered by the patient is a hoarse grunt as the air is forcibly driven through the flaccid glottis. With all this, the intelligence and memory, appetite and digestion, the functions of the bladder and rectum, are perfect, and, as a rule, the special senses, the movements of the eyes, the sensibility of the skin of the face and mucous membrane of the mouth, and motor and sensory power in the limbs are unaffected. Only in rare cases does the disease extend to higher centres, so as to produce deafness or ocular paralysis, or to affect the sensation of the face; and if motion of the limbs is affected it is by the lesions of progressive muscular atrophy or amyotrophic lateral sclerosis. The electrical excitability of the affected muscles is, in part at least, retained, but in advanced cases is much diminished. Erb says that the muscles of the chin, lips, and even the tongue show a marked reaction of degeneration, while the electric irritability of the nerves is normal or but slightly diminished. The reflexes are generally diminished, so that the palate, pharynx, or larynx may be irritated without exciting retching, vomiting, or coughing; but they sometimes persist till late in the disease, and Erb describes reflex contractions in the muscles of the chin and lips. Fever is absent, vaso-motor disturbances are not necessarily present, nor has glycosuria or albuminuria been observed as part of the disease. Occasionally, towards the end, the pulse becomes very rapid (140 to 160). Death takes place by exhaustion from inanition, by choking, by dyspnœa or sudden cardiac failure, or by bronchitis, pneumonia, or gangrene set up by the inhalation of particles of food.

Pathology.—To the naked eye the medulla oblongata may show but little, or there may be some want of symmetry, or slight shrinking; or, on section, discoloration or blurring of the outlines. Changes are more obvious in the nerve-roots proceeding from the medulla; those of the hypoglossal and facial, the vagus and accessorius, are grey in colour and shrunken, and the microscope shows that a number of fibres are atrophied and degenerated. Microscopic examination of the corresponding nuclei reveals degeneration and atrophy, or complete disappearance of the nerve-cells, some increase of the neuroglia, and thickening of the vessel-walls. These changes are most marked in the hypoglossal nucleus and the lower part of the facial nucleus, and then in the vago-accessorius nucleus. Less commonly the glosso-pharyngeal nucleus may be affected, and rarely the nucleus of the sixth nerve, and that of the motor division of the fifth.

The atrophied muscles present appearances identical with those seen in progressive muscular atrophy.

Diagnosis.—This is generally quite easy, from the chronic course and the limitation to the bulbar nerves specified. Tumours growing in or compressing the medulla are mostly accompanied by other symptoms, such as headache, noises in the ears, deafness, sickness, or convulsions. Bilateral lesions situated more centrally (*i.e.*, in the motor tracts nearer the cortex of the brain) may cause paralysis of the same nerves, but the symptoms on the two sides may not run parallel, and there will be no atrophy, electrical changes, or loss of reflexes, showing that the nerve-nuclei are intact; further, the limbs will be probably paralysed.

Prognosis.—This is absolutely unfavourable.

Treatment.—Drugs are of no value in controlling this disease. The important thing is to secure proper nutrition, and to prevent the inhalation of particles into the lungs. It may become necessary to feed the patient by an india-rubber tube, which the patient may himself pass down the pharynx into the stomach, and liquid food may then be poured into a funnel connected with the free end of the tube. Sometimes, in the early stage, solid food in large boluses can be more easily swallowed than liquid, being less liable to pass into the larynx or nares. Galvanism has been recommended, applied both to the seat of the disease and to the wasting muscles. The influence in the former situation—reached by placing one pole on each mastoid process—must be very doubtful; but the muscles of the lips and tongue may be galvanised, and the act of swallowing may be assisted by galvanising with the anode on the nape of the neck and the kathode on the side of the larynx. Atropine has been given to lessen salivation. The general health of the patient must be, as far as possible, maintained.

ACUTE BULBAR PARALYSIS.

Contrasting with the chronic progressive form, there occur, occasionally, cases in which the symptoms of bulbar paralysis come on suddenly, or at least rapidly, as a result of *hæmorrhage*, *embolism*, or *acute inflammation* of the medulla oblongata. In addition to the rapidity of the actual onset, they differ from the chronic cases in the greater frequency of premonitory symptoms, in the irregularity of the symptoms, and in the accompanying paralysis of the limbs; since the lesions are not necessarily restricted to motor nerve-nuclei as in the "progressive" cases, but are more or less indiscriminate, affecting the motor and sensory tracts as well.

Thus with paralysis of the tongue, difficulty of articulation, and inability to swallow, there may be paralysis of all four limbs. The occurrence of the lesion on one side will produce a more or less unilateral distribution of the symptoms. On the other hand, crossed paralysis may take place; for instance, paralysis of the arm on one side, and of the leg on the opposite, from a lesion in one half of the medulla affecting the lowest pyramidal fibres going to the opposite limb just before their decussation and the highest fibres coming to the same side just after their decussation. Or a hæmorrhage situated higher in the medulla may damage the facial nerve-fibres or nucleus on the same side, and the pyramidal tract of the same side, before its decussation to the opposite, producing a crossed hemiplegia such as results from lesions of the pons Varolii (*see* p. 273). Sometimes also there is severe respiratory disturbance, or rapid and irregular pulse, or vaso-motor derangement, shown by rise of temperature. Albumen and sugar have been noticed in the urine. There may, however, be little time for the observation of such symptoms, as, especially in hæmorrhage, the patient may fall down suddenly, with or without cry, and death may take place at once. In other cases there are headache, vomiting, noises in the ears, and epileptiform convulsions. In fatal cases the temperature sometimes rises to 107° F. or higher.

Pathology.—Bulbar hæmorrhage is more frequent than spinal hæmorrhage, less so than cerebral hæmorrhage. It occurs under similar circumstances to the latter. The same may be said of embolism or thrombosis of the arteries of the medulla oblongata; here, however, the distribution of the vessels becomes of interest, since, according to Duret, the nuclei of the hypoglossal and accessory nerves are supplied by the anterior spinal and vertebral arteries; those of the vagus, glosso-pharyngeal, and auditory nerves by branches of the upper end of the vertebral arteries; and the nuclei of the facial, trigeminal, and three oculo-motors by branches of the basilar. These anatomical associations may serve sometimes to distinguish vascular obstruction from hæmorrhage, otherwise not always easy to discriminate. Acute bulbar myelitis is usually less rapid in its occurrence than the other lesions; vertigo, headaches, pain in the muscles of the back, may precede the more obvious bulbar symptoms. The limbs may be paralysed from implication of the pyramidal tracts. The temperature is sometimes raised, and the pulse is mostly rapid. Death takes place in from four days to two or three weeks.

COMPRESSION AND TUMOURS OF THE MEDULLA.

The medulla oblongata may be slowly compressed as a result of caries of the occipital bone, or by alterations of its shape, by enlargement of the odontoid process, by tumours such as gumma of the dura mater, growths on the choroid plexus, aneurysms on the vertebral or basilar arteries, and lastly, perhaps most frequently, by tumours of the cerebellum.

The rare occurrence of disease and rupture of the transverse ligament is followed by sudden and fatal compression of the medulla by the odontoid process. Tumours in the medulla oblongata are comparatively rare; they include tubercular masses, glioma, glio-sarcoma, myxoma, and fibroma.

Symptoms.—In compression, the symptoms characteristic of bulbar lesions may be preceded by those of irritation, such as pain in the distribution of the fifth nerve, and twitchings in muscles supplied by the facial. Convulsions, vomiting, hiccup, and dizziness may also be present; and, later, the special lesions of the bulbar nerves, and probably weakness in the limbs. The symptoms may begin on one side and spread to the other. Tumours in the substance of the medulla are not accompanied by irritative symptoms; but headache, vomiting, and convulsions may occur. Probably optic neuritis is only observed in the case of tumours sufficiently large to implicate the pons Varolii.

DISEASES OF THE BRAIN.

What has been said of the nervous system in general is especially true of the brain—namely, that the symptoms of disease are largely determined by the locality of the lesions, and to a much less extent by their nature. A knowledge, therefore, of the localisation of the functions of the brain is eminently desirable in the study of its diseases, and some remarks on this subject, with an account of the result of lesions, of whatever kind they may be, upon the different parts of the brain, must precede the description of the diseases which produce them.

Motor Centres and Tract.—The greatest importance and interest attach to the position of the motor centres. It has been shown that electrical irritation of a certain area of the grey matter on the surface of the brain produces movements of different parts of the body, according to the spot irritated; and these experimental results are confirmed by pathological evidence, certain (irritative) lesions causing convulsions, and other (destructive) lesions causing paralysis, in the corresponding parts. In accordance with this is the fact that the largest pyramidal nerve-cells (upper motor neurons) are found exclusively in the grey matter of this area. This *motor area* comprehends the convolutions about the fissure of Rolando, the ascending frontal, the ascending parietal (central convolutions of Continental authors), the superior parietal lobule, and the posterior part of the marginal convolution, which corresponds to the upper ends of the central convolutions (para-central lobule). In this area, the centres for movements of the *face* lie lowest, or farthest from the middle line, occupying the lower and middle portion of the ascending frontal, posterior extremity of the third frontal, and the lower end of the ascending parietal; the centres for the *arm* lie next, nearer to the middle line, in the upper part of the ascending frontal convolution, the posterior end of the superior frontal, and the middle of the ascending parietal convolution; the centres for the *leg* occupy the upper ends of both ascending frontal and ascending parietal convolutions, and the superior parietal lobule close to the middle line; and lastly, the posterior part of the marginal convolution is found to correspond mainly to the muscles of the *scapula*, *trunk*, and *hip-joint*. Fibres from these several convolutions gradually converge through the white matter of the centrum ovale to the base of the brain, and there pass between the lenticular nucleus and the optic thalamus, occupying the genu and the anterior two-thirds of the posterior limb of the internal capsule. Thence the pyramidal fibres pass into the lower part of the crus cerebri, at first somewhat on the outer side, and then in the middle; and they can be traced through the pons Varolii into the medulla oblongata, where the *decussation* takes place, the greater part of each pyramidal tract crossing to the opposite lateral column, a small portion remaining on the same side at the inner margin of the anterior column, forming the column of Türck, the fibres of which successively decussate lower down in the cord. The pyramidal fibres, the axis-cylinders of the upper motor neurons, ultimately terminate in the anterior cornua, by arborisations about the cell-bodies of the lower neurons (p. 164).

A sufficiently large destructive lesion of any part of the pyramidal fibres from the motor centres to the pons Varolii causes *hemiplegia*, or paralysis of the face, arm, and leg, on the *opposite* side of the body. This is a common result of a hæmorrhage into the internal capsule; but disease above this, where the fibres are spread over a wider area, may lead to a more limited paralysis. Extensive lesions of the cortex, such as suppurative meningitis, or meningeal hæmorrhage, will also cause complete hemiplegia, but smaller lesions may occupy only particular centres, and give rise to the forms of paralysis called *monoplegia*. Thus a disease of the facial centres results in paralysis on the opposite side of the facial muscles alone, *facial monoplegia*; and in a corresponding manner there may be a *brachial monoplegia*, or a *crural monoplegia*. If the adjacent centres of the face and arm are involved together, *brachio-facial monoplegia* is the result, and if those of the arm and leg together, a *brachio-crural monoplegia*. But the centres for the face and leg can scarcely be affected at the same time, without those for the arm, which lie between them, being involved, and, as a fact, a *cruro-facial monoplegia* has never been recorded.

Sensory Tract.—The relations of the sensory neurons in the brain have been mentioned (p. 165). Much of the sensory tract passes through the posterior third of the posterior limb of the internal capsule, since this part has been found injured in cases of anæsthesia of the opposite half of the body (*hemianæsthesia*). The terminal arborisations of the upper sensory neurons come into indirect relation with the cortical motor cells; and thus probably sensation is represented by much the same convolutions as those which form the motor areas.

Frontal Convolutions.—The posterior extremities of the upper two frontal convolutions, adjacent to the ascending frontal, contain, according to Ferrier, centres for rotation of the head and eyes; the posterior extremity of the third frontal convolution is connected on the left side with speech movements, so that a lesion there situated causes motor aphasia (p. 279). The remaining portion of the frontal convolution seems to be related to the intellectual functions.

Superior Temporo-sphenoidal Convolution.—This contains the centre for hearing, and lesions of certain parts of it produce the condition known as word-deafness, in which words are heard as sounds, but their meaning is not understood (p. 280).

Occipital Convolutions.—These contain cortical centres for vision, and their destruction on one side causes hemianopia of the opposite. Visual centres have also been placed in the angular gyrus and supra-marginal lobule; lesions of these produce word-blindness, in which words are seen but not understood (p. 280).

Optic Thalamus.—The posterior part of this grey mass contains the central ends of some optic nerve-fibres, and lesions may produce complete hemianopia. Disease of the optic thalamus has also been present in cases where hemiplegia has been followed by choreic or other disorders of movement.

Internal Capsule.—This is the layer of white matter lying, in front, between the lenticular nucleus and the anterior end of the caudate nucleus; behind, between the lenticular nucleus and the optic thalamus. These two portions are not in one plane, but join at an obtuse angle, opening outwards. The anterior portion is stated by Brissaud to contain fibres connected with the intellectual functions; at the angle (or *genu*) pass fibres connected with the movements of the eye, the head, the tongue, and the mouth; and through the posterior limb, the remaining pyramidal fibres (anterior two-thirds), and the sensory fibres (posterior third). Near this posterior third it is stated that fibres run, an injury to which is followed by post-hemiplegic chorea (p. 276). The pyramidal fibres have the following arrangement from before backwards. Next to the fibres for the mouth are those for movements of the shoulder, and then successively those for the elbow, hand, abdomen, hip, knee, and foot.

Cerebellum.—Lesions of the hemispheres are not always accompanied by symptoms unless they are of such a nature and size as to press upon the subjacent pyramids of the medulla oblongata, or such as to involve the median lobe. In this latter case occurs a characteristic form of ataxy (p. 310), vertigo, and sometimes convulsions of tetanic and opisthotonic character.

Pons Varolii.—This portion of the brain contains the pyramidal tracts, and the nuclei of the fifth, sixth, and seventh nerves. Large central lesions may paralyse all four limbs from the proximity of the two tracts to the middle line. A one-sided lesion in the upper part produces hemiplegia of the ordinary type on the opposite side of the body; but a lesion in the lower part, while involving the same pyramidal fibres for the arm and leg, is below the facial fibres for the opposite side, and destroys the facial nerve-roots of its own side. There is then produced a variety of hemiplegia known as *crossed hemiplegia*, the face being paralysed on the side of the lesion, the arm and leg on the opposite side.

HEMIPLEGIA.

Before dealing with the diseases of the brain it is desirable to describe in more detail the form of paralysis known as hemiplegia.

Though this term seems to imply paralysis of one half of the body, as a fact some muscles are not paralysed in ordinary cases of hemiplegia, and of those that are paralysed some suffer much more than others. Paralysis is most marked in the arm, leg, and face, on the side opposite to that of the lesion; the muscles of the trunk, chest, and abdomen are but little affected, and the ocular muscles not at all.

Even in the face, arm, and leg, many differences exist. In severe cases the arm and leg may be completely motionless, but in milder cases the leg is less paralysed, and in course of recovery the leg nearly always gets better first. The facial muscles are never paralysed to the same extent as they are in a lesion of the nucleus or trunk of the facial nerve (*see* p. 195); the upper half of the face is always less affected. Thus, the eye can always be shut, and the forehead wrinkled, though it may be obvious that the frontalis and the orbicularis are not so strong as those on the opposite side. In the lower part of the face the paralysis is marked; if the patient is told to smile, or to show the teeth, the angle of the mouth is drawn up on the healthy side, and on the paralysed side it remains unmoved or is drawn inwards. Sometimes there is a slight degree of paralysis of the tongue shown by the tip being turned, during protrusion, to the paralysed side by the unbalanced action of the healthy genio-hyoid and genio-hyoglossus muscles. In examining this point, the asymmetry due to the facial paralysis must be remembered, and the position of the tongue must be determined by its relation to the teeth rather than to the lips. Sometimes the chest on the paralysed side may be seen to expand less freely than on the other, and the weakness of the abdominal muscles on the same side may be shown when the patient coughs; but any paralysis of the spinal muscles is not commonly observed, and the movements of the eyeball are perfect. The explanation commonly adopted for these variations of paralysis, under apparently similar anatomical conditions, is that suggested by Broadbent. It is first to be observed that the parts that are least paralysed, or not paralysed at all, are those which rarely or never act independently of their fellows on the opposite side; whereas the parts that are most paralysed are much more independent, and may be capable of performing acts which the corresponding muscles on the opposite side are unequal to. As extreme instances may be mentioned the eyes, of which one never moves except in association with the other; and their muscles are not affected. In contrast with these are the hands, of which the right may be able to do things the left cannot, and *vice versâ*; and these parts are most affected. Broadbent's theory supposes that in the case of the muscles most commonly associated together, the commissural fibres between their nerve-nuclei become functionally active, so that in the event of a lesion preventing one, say a right side nucleus, from receiving stimuli from the left brain, it may be stimulated from the right brain by impulses passing first to the left side nucleus, and then by the commissure to the right side nucleus. On the other hand, if in the case of the less associated muscles the commissure remains functionally inactive, such a transference would not take place, and the right side nucleus would remain completely cut off from the cortical centres. It may be doubtful at what exact point the functional communication between the two sides of the nervous centres takes place; but in any case the bilaterally associated muscles can be stimulated by both hemispheres, whereas the muscles acting independently are excited by the opposite hemisphere only.

Causes of Hemiplegia.—This form of paralysis may arise from any lesion involving the pyramidal tract from the cortex to the upper part of the pons Varolii. Its most frequent causes are cerebral hæmorrhage, cerebral embolism, and thrombosis; and others are injury to the surface of the brain, meningitis, whether suppurative or tubercular, encephalitis, tumours, and abscess.

Associated Conditions.—The duration of hemiplegia is very variable. It may get completely well in the course of a few weeks, the power of movement being gradually restored in the face first, and the leg afterwards, so that the patient can walk about, while the arm is still useless. Sometimes, however, recovery progresses up to a certain point during the first two or three months, until a stage is reached beyond which no improvement takes place. The paralysis is then generally accompanied by a rigidity or contracture of the muscles, which gradually develops during the first few weeks. The fingers become flexed into the palm of the hand, and the elbow is slightly bent; any attempt to overcome the flexion is resisted, and causes considerable pain. In the lower extremity the knee is only slightly flexed, and the foot is often extended at the ankle. With this so-called *late* rigidity—to contrast it with the rigidity of the apoplectic state described below (p. 283)—the deep reflexes are increased, the knee-jerk is greater, and ankle-clonus is readily obtained. The muscles may lose a little of their bulk from disuse, but are not degenerated, and the electrical reactions, faradic and galvanic, remain normal. This condition is precisely similar to what is seen in lesions of the pyramidal fibres (upper neurons) in the spinal cord, *i.e.*, a spastic paralysis. In the same way the rigidity and increased reflexes have been attributed to the secondary degeneration of the lateral tract; but the increased reflexes, at least, are present from an early stage of the paralysis, before degeneration can have become well established. More rarely there occur in the partially-paralysed muscles other disorders of movement, of which the most important are *athetosis* and *post-hemiplegic chorea.*

In *athetosis* there is a constant involuntary slow movement of the fingers, which are abducted, adducted, flexed, and extended in the most irregular way. Similar, but generally less extensive, movements may affect the arms and the toes. In *post-hemiplegic chorea* the movements are more violent, jerking and ataxic, and affect the arm and leg as much as the fingers and toes. Both contractures and post-hemiplegic movements are very intractable. In many cases of athetosis an old lesion of the corpus striatum or optic thalamus has been found.

Anæsthesia of any extent is not common with hemiplegia. If it occurs with the onset of the attack it generally passes off within a few days; but occasionally a complete *hemianæsthesia* is present. This is on the same side of the body as the paralysis—that is, the side opposite to the lesion—and it affects equally the face, arm, leg, and trunk up to the middle line of the body. The special senses are affected at the same time, the patient being unable to smell, taste, hear, or see on that side of the body. The affection of sight is a real blindness of one eye, or amblyopia, and not loss of one half of the visual field, or hemianopia (*see* p. 185).

Conjugate deviation, or forcible rotation of the head or eyes to one side, is occasionally associated with the sudden or apoplectic onset of hemiplegia: it may continue after consciousness has returned; but it generally subsides after a few days or a week. Ferrier has found that there is a region of the cortex, situate in the *frontal* lobe, irritation of which causes deviation of the head and eyes; but pathological results show that conjugate deviation is not restricted to lesions of one locality alone. There is, however, an important connection between the position and the nature of the lesion and the side to which the deviation occurs When a lesion of the *left* side of the brain occurs, giving rise t *right* hemiplegia, the eyes and head are turned to the *left* side that is, the eyes are turned *away from the paralysed side*, or th patient is said to *look towards his lesion.* But if, as a result o this lesion, there should occur convulsions in the paralysed limb or if a cerebral lesion of any kind causes convulsions on t opposite side with rotation of the head, neck, and eyes, t rotation will be *towards* the convulsed side—that is, the patie will appear to *look away from his lesion.* And, similarly, w right-sided lesions, there will be left-sided paralysis with rig sided deviation, or left-sided convulsion with left-sided deviati

Always, then, in *cerebral* lesions, deviation with paralysis is away from the paralysed side, deviation with convulsion is towards the convulsed side. So far as the nerve mechanism is concerned, the following explanation of the phenomena was given by Ross: For purposes of vision on any one side of the body, the external rectus of that side receives the first stimulus and the internal rectus of the opposite side is at the same time innervated, not directly from its cortical centre, but by commissural fibres between the nucleus of the third nerve and that of the sixth. With increased stimulus to see to the right, the rotator muscles of the head and neck (deep muscles on the right side, and sternomastoid on the *left* side) are brought into action, being also innervated by commissural fibres between the sixth nerve-nucleus and their own. Thus a cortical or supra-nuclear lesion on one side stimulating the centre of the opposite sixth nerve will cause convulsive deviation of the opposite eye to the opposite side, away from the lesion; and the other associated muscles will follow suit. On the other hand, a destructive cortical or supra-nuclear lesion on one side will paralyse the opposite sixth nerve and with it the nerve to the internal rectus of the same side as the lesion, and the nerves to the muscles which rotate to the opposite side; and hence the rotation of these parts by the action of antagonistic muscles *towards* the lesion. This explanation seems to me much less satisfactory as regards the paralytic deviation than as regards the deviation with convulsion. Spasm of its antagonist is not the necessary, nor even the usual, immediate result of paralysis of any muscle; and in the paralytic cases, the deviation of the head is forcible and spasmodic, and it resists all ordinary efforts to replace it in a median position.

Lesions in the *pons Varolii* may also cause deviation, but the results are the converse of the above; because at this level a lesion which damages the pyramidal fibres corresponding to the opposite arm and leg will involve the sixth nerve (and nucleus) to the external rectus of the damaged side. With a destroying lesion the deviation is towards the paralysed side and away from the lesion; with a convulsing lesion the deviation is away from the convulsed side and towards the lesion.

Another condition frequently associated with hemiplegia is a form of loss of speech, called *aphasia*. It is described below.

Hemiplegia commencing in early childhood presents some special features, and it will be separately described with the subject of Encephalitis (*see* Infantile Hemiplegia).

APHASIA.

The term *aphasia* means loss of speech from a cerebral lesion and must be distinguished—

(1) From *aphonia*, or voicelessness, which is due to failure o the laryngeal muscles, and is not indeed a loss of speech, sinc words can be uttered by whispering;

(2) From *anarthria*, or defect of articulation, which is due t imperfect action of the muscles of the lips and tongue, consequen upon lesions of the medulla oblongata or the nerves proceedin thence (bulbar paralysis);

(3) From purely *mental* aberrations independent of demonstrabl lesion of the cerebral centres.

With rare exceptions aphasia is due to a lesion on the left side of the brain, and consequently, if it is associated with hemiplegia, it is with a *right hemiplegia* and not with a left hemiplegia. The only explanation of this is that the left side of the brain is alone or chiefly educated for speech processes; and this view receives support from the rare cases of aphasia associated with left hemiplegia, in which it has often been found that the persons were left-handed. It is thus suggested that the side of the brain which is educated for the most extensive use of its associated opposite arm also develops the functional activity of its speech-centres, while those of the opposite side are comparatively inactive, though there is reason to believe that they do take some share in speech-processes. In aphasia, then, the muscles of the lips are used perfectly so far as the utterance of any letters or even of syllables is concerned. The words which the patient can speak are clear, distinct, and natural, or if words and syllables are mixed unintelligibly, it is obviously from imperfection of the higher centres or from want of perfect automatism in them, rather than in the action of the muscles of articulation. There is none of the blurred utterance or thick speech of bulbar paralysis, general paralysis, or alcoholism. But the possible defects are very numerous, and are as follows:—

The patient may be able to utter no word at all; or he can only say a few words, such as "yes" or "no," or give his name, or he may have the use of some half-dozen words, which he gives in answer to every question. He is unable to give names to any object shown him. In some cases, if the patient is told the name, he appears not to recognise it, and certainly cannot repeat it; in other cases he recognises it at once and repeats it. Some patients are utterly unconscious of the unfitness of the words they use for the ideas they seem to want to express; others perceive at once their mistakes, and manifest, as a rule, considerable annoyance thereat. If aphasia coexists with right hemiplegia it is not surprising that the patient should be unable to write; but if, as may happen, aphasia occurs alone, or a slight hemiplegia quickly recovers, it is observed that the patient with aphasia is also unable to write intelligibly or at all, a condition which is called *agraphia*.

The above defects may be conveniently arranged for investigation of any case in the following order, assuming, of course, that the patient can see and is not totally deaf. He may be unable (*a*) to hear words spoken; (*b*) to understand words spoken; (*c*) to see words written and printed; (*d*) to understand words written and printed; (*e*) to speak from memory; (*f*) to repeat words; (*g*) to read aloud, *i.e.*, to speak from sight; (*h*) to write from memory; (*i*) to write from dictation, *i.e.*, from words heard; (*k*) to write from a copy, *i.e.*, from words seen.

Speech depends not only upon perfect co-ordinating and motor (outgoing) processes, but also very largely upon the functions by which the materials for speech are supplied, and these are the senses of hearing and of sight.

It is sufficient here to point out that the child learns to speak by attempting to imitate words which it first hears, and that subsequently the process is very much aided by printed and written words and by objects which are seen.

The nerve apparatus concerned consist of the following parts:—(1) The auditory speech centre, in the upper extremity of the first left temporo-sphenoidal convolution; (2) the visual speech centre, in the left angular gyrus and supra-marginal gyrus; (3) the motor speech centre, in the posterior part of the third left frontal convolution, or Broca's convolution; (4) the motor writing centre, probably in the posterior part of the second left frontal convolution, related to the muscles of the hand; (5) commissural fibres between these. An ideational or psychical centre (6), in commissural connection especially with (1) and (3), is assumed by some authors, but denied by others.

Lesions of the first four centres and of the commissural fibres will cause some form of aphasia or agraphia. If the motor speech centre is destroyed there is *motor aphasia*. If either the auditory or the visual speech centre is destroyed there is *sensory aphasia*. A lesion may be large enough to involve two or more of these centres, or commissural connections rather than centres may be injured, so that various combinations of aphasic conditions may result in different instances.

Motor Aphasia.—The patient is speechless, or has but few words at command. There are often words like "yes" or "no," or interjections, or words used emotionally, as oaths; he can understand what is said to him, his sensory centres being unaffected; but he cannot repeat; he is perfectly aware of any errors that he makes, if he says anything at all. Thus he is deficient in respect of *e*, *f*, and *g*. In many cases of motor aphasia the patient has also other defects. Thus he may have *alexia*, or inability to read so as to understand (*d*), and this has been thought to result because the motor speech processes are so much employed in the act of reading. And he may have *agraphia* (*h*, *i*, *k*); sometimes he may be able to write, but not to copy. But it has been shown that motor aphasia can occur without either alexia or agraphia: alexia when present is probably due to a co-existing lesion of the auditory word-centre, and agraphia to a lesion of the centre above mentioned as probable.

Sensory Aphasia.—This is associated with one or other of two functional defects—*word-deafness*, due to lesion of the auditory speech centre; and *word-blindness*, due to lesion of the visual speech centre. In word-deafness the patient can hear ordinary sounds, but cannot recognise and understand words spoken to him, cannot repeat words, and cannot write from dictation. As the auditory word-images are essential both for perfect written and spoken matter, he speaks, reads aloud, and writes imperfectly, using wrong words and syllables—*paraphrasia* and *paragraphia*. Thus he is defective in *a*, *b*, *d*, *f*, *g*, *i*, *k*.

In word-blindness the patient cannot see or understand words printed or written, cannot read aloud, and cannot write spontaneously from dictation or copy, as writing is stimulated directly from the visual centre, but he may be able to talk well. His defects are *c*, *d*, *g*, *h*, *i*, *k*.

In a variety of word-blindness (*sub-cortical alexia*) the lesion is below the cortex, cutting through commissural fibres only. The patient can then write voluntarily and from dictation. In these cases also, or in any lesions of the visual speech-centre deep enough to involve the optic radiations of Gratiolet, the connections of the centre with the retina are injured, and the patient has hemianopia of the side opposite to the lesion.

Two other conditions may be associated with sensory aphasia: they are *mind-deafness*, or inability to recognise the meaning of any kind of sound; and *mind-blindness*, or inability to recognise the meaning or use of any kind of object.

Recovery, or at any rate improvement, in speech may result from education of the opposite hemisphere—a very slow process—or from stimulation of the motor centre by the opposite hemisphere in cases where the lesion is in the motor path, below the speech-centre itself, and where the callosal fibres connecting the speech-centre with the opposite side are untouched.

Besides the organic lesions, such as embolic softening and hæmorrhage, which more commonly produce aphasia, functional disturbances may cause it, such as migraine and right-sided convulsions. Hysteria more often leads to aphonia from laryngeal failure.

CEREBRAL HÆMORRHAGE.

In dealing with diseases of the brain the greatest prominence must be given to diseases of the vessels of the brain, which are responsible for the majority of cases of cerebral paralysis. Rupture of the vessels, with escape of blood (*hæmorrhage*) into the brain, and obstruction of the arteries by *embolism* and by *thrombosis*, are the forms of vascular lesion that have to be considered.

Ætiology.—Cerebral hæmorrhage occurs more frequently in men than in women, and more in advanced life than in youth. Thus it occurs, but is very infrequent, below the age of thirty, and has, indeed, occurred in one as young as nine; but something like four-fifths of the cases occur after the age of forty. A large proportion have granular kidneys and hypertrophied hearts; the arteries are generally at the same time thickened or atheromatous; and frequently the actual cause of hæmorrhage is the rupture of minute (miliary) aneurysms, which are found on the branches of the cerebral arteries. Alcohol, gout, and syphilis have their share in the production of these arterial lesions. Hæmorrhage is sometimes associated with heart disease and endocarditis, and this is especially the case when it occurs in quite young people. Probably in these instances an embolism has been the first lesion, around which the artery has softened, and then it has ruptured; or the artery has yielded so as to form an aneurysm which has subsequently burst—a process similar to what will be described as occurring in the hæmorrhage from the lung in phthisis. The comparatively slight support that the walls of arteries receive from the soft brain-tissue may be a reason why they rupture so much more often than vessels in other parts of the body. Hæmorrhages often occur in the substance of tumours of the brain, and they may be so large as to render their source uncertain. Lastly, there may be smaller hæmorrhages in conditions of general tendency to bleeding (scurvy, purpura), and as a local result after ligature of the common carotid artery.

Seats of Hæmorrhage.—Scarcely any part of the brain is exempt from the risk of hæmorrhage, but it is much more frequent at the base in the neighbourhood of the corpus striatum and optic thalamus, which are mainly supplied by the branches of the middle cerebral artery. One large branch runs upwards on the outer side of the lenticular nucleus, and has been called by Charcot the "artery of cerebral hæmorrhage," from the frequency with which it ruptures. Vessels may also burst in the lateral ventricles (*ventricular hæmorrhage*), or on the surface of the brain (*meningeal hæmorrhage*); but when blood is found in these situations, it has often proceeded from a hæmorrhage primarily in the cerebral substance.

Anatomical Changes.—Under different circumstances the blood effused may be small in quantity, or amount to several ounces. In the latter case it tears up the brain-tissue, destroying, for instance, the great ganglia, and the internal capsule, and extending thence into the centrum ovale; or it may burst through the optic thalamus or caudate nucleus into the lateral ventricle. Thence the blood flows by the aqueduct of Sylvius into the fourth ventricle. Such cases are rapidly fatal, and *post-mortem* examination reveals a mass of black clot, filling the ventricle, and occupying much of the hemisphere, surrounded by brain-tissue, which is ragged, and discoloured by blood. The pressure exerted by the clot is shown by one, or even both, hemispheres being enlarged, with flattened convolutions, and closed sulci. In cases which have lasted a few days, there is the same black-red clot, and the brain-tissue around is soft and discoloured yellow, from absorption of hæmoglobin. In later stages, the clot becomes brown, or brownish-yellow, consisting of disintegrated blood and nerve-tissue; and the surrounding tissue is frequently softened (white softening), and contains granule-corpuscles. Finally, in patients that survive, the blood becomes absorbed, and leaves a tawny or orange-coloured spot, in which crystals of hæmatoidin can be found; or a cyst may remain, containing serous fluid; or a distinct, tough, fibrous scar, discoloured also by the remains of blood-pigment.

Secondary Degeneration.—Permanent lesions of the pyramidal tract, or of the cortical motor area, are followed by descending secondary degenerations, like those which occur in disease of the spinal cord. Such degenerations follow the course of the pyramidal fibres below the lesion; thus, a lesion of the internal capsule (and it is to be observed that lesions of the corpus striatum and optic thalamus alone are not followed by secondary degeneration) causes this change to take place in the middle third of the crus cerebri, in the anterior part of the pons, in the pyramid of the medulla oblongata on the same side, in the column of Türck, also on the same side, but in the posterior part of the lateral column of the spinal cord for its whole length on the opposite side.

Course and Symptoms.—*Apoplexy.*—Cerebral hæmorrhage may be preceded for days or weeks by occasional giddiness, numbness or twitching of the fingers, or headaches; but these are not so much indications of the severe attack to come, as evidences of existing disease of vessels, and perhaps due themselves to slight hæmorrhages. On the other hand, it may come on without any warning whatever. Sometimes, also, it seems attributable to a definite cause, such as a muscular effort, violent coughing, or straining at stool, but at others it occurs when the patient is perfectly quiet, or even during sleep. In numerous cases cerebral hæmorrhage causes *apoplexy*—that is, the patient is struck down suddenly unconscious or quickly becomes so; and the association between hæmorrhage and apoplexy has been so close that the term apoplexy has been applied to the actual bleeding as well as to the special symptom which it causes, not only in the brain, but also in other organs: for instance, hæmorrhage in the lung has been described as pulmonary apoplexy. But this is clearly wrong, and the term apoplexy ought to be restricted to the idea of sudden, or at least rapid, loss of consciousness and power. As a fact, it is rarely that a patient is absolutely struck down and unconsciousness in a moment; but it does happen, and patients have died in five or ten minutes from the first symptom. More often the symptoms come on slowly. The patient is seized with intense pain in the head, becomes faint or slightly collapsed, may be sick or have a slight convulsion; and then, after half an hour or so, gradually sinks into a condition of coma. This order of events has been called *ingravescent apoplexy*. Or the first symptoms may show themselves in the motor system; the patient mumbles in his speech, or his arm falls powerless, and he gradually droops over to one side, falling if not supported, and then lapses by degrees into coma. Or the coma may be developed in a few hours through stages of increasing drowsiness. Occasionally the attack begins with convulsions, or vomiting occurs as an early symptom. Lastly, in cases where the patient is found by the friends alone, or picked up in the streets unconscious, or is unable to be roused in the morning from sleep, it is impossible to say what the onset has been. But undoubtedly cerebral hæmorrhage may occur without apoplexy; a very slight bleeding into the motor tract alone may give rise to paralysis without loss of consciousness.

The patient suffering from hæmorrhagic coma lies completely unconscious, and cannot be roused by shouting or any form of stimulation of his skin. The face is flushed, the pulse is full and tense, the breathing is *stertorous*, a loud snoring noise being made in consequence of the palate or tongue falling back and impeding the passage of air into the chest. The condition of the limbs varies: both legs and arms may be quite flaccid, falling at once when raised; or it may be obvious that the leg and arm on one side are more flaccid than those on the other. The muscles of the face share in the paralysis, and the cheeks are puffed out and sucked in with the processes of respiration; this may also occur only on one side. Sometimes, however, the limbs of one or both sides are in a condition of rigidity, the muscles contracted and resisting extension or flexion. The deep reflexes are commonly increased, and the skin reflexes are absent. The pupils are variable; they are sometimes contracted, at others dilated or unequal. Conjugate deviation may occur. The temperature shows a slight fall, which may continue till death, or if life is prolonged it rises a little above the normal. Sugar and albumen are occasionally found in the urine, from pressure on the medulla oblongata. In very severe cases, the pulse and breathing are rapid, there is profuse sweating, and intense flushing of the face and skin generally; then after a time, two or three hours or more, the patient becomes livid, râles occur in the larger bronchi and trachea, the pulse gets weaker, the breathing slower, and finally death takes place. The fatal termination may, however, be delayed for several days, during which the lungs are very apt to suffer from œdematous pneumonia; and the occasional passage of particles of food or fluids through the glottis perhaps contributes to the inflammation of these organs. In more favourable cases, the patient lies simply comatose, with but little disturbance of his pulse or respiration, and gradually regains his senses in the course of a few hours or two or three days. In a large proportion of cases, the patient is then found to be suffering from *hemiplegia*, which may itself slowly recover or be permanent (*see* p. 274).

Diagnosis.—This has to be made from embolism, from uræmia, from alcoholic intoxication, and other conditions; it will be best considered after cerebral embolism has been described.

Prognosis.—This is generally unfavourable in proportion to the extent or severity of the first symptoms. That apoplexy is often fatal is well known, and death may take place at different intervals after the onset. The cases are grave in which the coma is profound, with much stertor, flushed or congested face, full bounding pulse, and complete relaxation of all the limbs.

If, after recovery from the first coma, headache continues, and the patient again becomes drowsy, the result is likely to be serious, and probably inflammatory changes are taking place about the clot, or in the area of softening.

Treatment.—In an attack of apoplexy, the patient should be in the recumbent position, with the head and shoulders slightly raised. Stertor is often diminished by placing him on one side, and this is further beneficial by allowing the full play of at least one lung—viz., that which is uppermost. An ice-bag should be applied to the head. If the bowels are known to be confined, or not recently opened, a drop of croton oil or a few grains of calomel may be placed on the tongue, or an enema of castor oil or turpentine may be administered. If the coma continues the catheter may be required to empty the bladder. Venesection is rarely carried out now, and is not generally desirable, though formerly it was the universal practice. By some writers it is considered justifiable where there are signs of high arterial tension, with deep congestion of the face, strong pulsation of the carotids, and full slow pulse; and Fagge suggested that it might be right in cases of ingravescent apoplexy, when, after the first shock or collapse, the circulation is beginning to re-establish itself, and thus a second hæmorrhage may possibly be avoided. Occasionally, rapid improvement under the use of bleeding, or of leeches applied to the temple, has been seen, but the nature of such cases must be doubtful. In later stages, pain in the head requires the continued application of ice, the patient should be carefully nursed to prevent the formation of bed-sores, and a water-bed may be needed. If hemiplegia occur without loss of consciousness, the patient should be kept quiet in bed, the bowels should be attended to, and a light diet of fish, milk, custard puddings, &c., should be enforced for some days or weeks. Neither here, nor in the cases beginning with coma, can the treatment of the paralysis be entered upon until all evidences of active mischief in the brain have subsided. Generally, after six weeks or two months the paralysed limbs may be gently faradised; but if headache, dizziness, or other unpleasant sensations are caused by this treatment, it must be delayed yet longer. As the muscles contract to faradism, this form of electricity may be employed, with the object of maintaining the muscles in the highest state of efficiency possible to them under the circumstances. If contractures show themselves, passive movements, frictions and faradisation of the antagonists of the contracting muscles should be employed. The possibility of a second attack should be remembered, and the patient should have always a light simple diet, should avoid alcohol in excess, take only moderate exercise, and keep from business or other mental worry as much as possible.

EMBOLISM AND THROMBOSIS OF CEREBRAL ARTERIES.

Pathology.—The usual cause of *embolism* is mitral or aortic endocarditis; the former is far more common. In either case particles of fibrin are detached from the surface of the valves, or in the case of a contracted mitral orifice, fibrin may be deposited in the dilated left auricle, and subsequently detached and carried into the cerebral vessels. The middle cerebral artery is more often obstructed than others, but the statement of most writers that the left is much more often plugged than the right is doubted by Fagge, who found in twenty-one cases, taken without selection, that the right artery was plugged in ten, the left only in eleven.

Thrombosis is most frequently caused by disease of the vessel wall, such as atheroma, by which the surface is roughened, and fibrin is consequently deposited. Syphilitic disease of the arteries produces considerable narrowing of their channels, and also invites thrombosis. In addition, thrombosis may occur from several conditions weakening the circulation, such as those resulting from enteric fever, typhus, cancer, phthisis, and other severe illnesses.

Embolism and thrombosis, by obstructing the circulation of the blood, alike lead to *softening* of the districts of the brain to which the vessels correspond, unless the vascular supply is maintained by means of anastomoses. These are not abundant in the case of the cerebral vessels, and, indeed, the vessels going to the central ganglia are really terminal vessels, while those going to the cortex of the brain do anastomose more or less. At least, this is true of the distribution of the middle cerebral artery—the vessel most often obstructed. A part of the brain in which softening has taken place has generally lost the smooth, glistening surface of a normal brain-section, is more opaque, or grey, or speckled; it breaks down readily under a stream of water; or it is milky, or diffluent. It shows under the microscope drops of myelin, portions of nerve-fibres, granule-corpuscles, and free fat-globules. It sometimes has a yellowish or brownish colour from blood-pigment; or minute extravasations may be present in cases of sudden obstruction, and a form of *red softening* results. In cases of rapid death after embolism, the brain substance may look perfectly healthy, as there has not been time for any changes visible to the naked eye to take place. Occasionally an embolus sets up inflammatory changes in its neighbourhood; sometimes it leads to aneurysm and cerebral hæmorrhage as already described (p. 281). Rarely actual infarcts are formed. The later stages of softening consist in the absorption of the disintegrated tissue, and the formation of a cyst; or, if the softening is small, a cicatrix may be produced.

Symptoms.—The symptoms of *embolism* are often precisely the same as those of hæmorrhage. Obstruction of a large vessel will cause sudden loss of consciousness, and death may take place soon after. In other cases, coma comes on more gradually, and may be preceded by pain in the head. When the patient comes out of his coma, he is often found to be paralysed on one side, and if the paralysis is on the right side, aphasia may be present. Since the softening occurs only in the areas supplied by the vessel beyond the seat of obstruction, the symptoms are more likely to correspond with the distribution of the artery than in hæmorrhage, where the extravasated blood ploughs up the brain with little discrimination. If the middle cerebral be obstructed near its origin, there will be hemiplegia of the opposite side; and if the lesion is on the left side, aphasia also, since this artery supplies the internal capsule, Broca's convolution, the greater part of the motor area of the cortex, the first and second temporal convolutions, and the angular gyrus. Perhaps more often than in hæmorrhage, embolism leads to sudden hemiplegia without loss of consciousness.

Thrombosis is usually less rapid in its effects, though with the same results—apoplexy and hemiplegia; but sometimes a sudden coma occurs, indistinguishable from that of hæmorrhage. There may be premonitory symptoms—headache, dizziness, loss of memory, with numbness or formication of an arm or leg, or of one side of the body. Senile forms of disease are frequently of this nature. The symptoms are aggravated from time to time by fresh lesions, and they are often accompanied by dementia.

Diagnosis of Cerebral Vascular Lesions.—This may be divided into two heads—the diagnosis of apoplexy from other conditions simulating it, and the diagnosis from one another of the different causes of apoplexy or hemiplegia.

(1) In the former the history is of great importance. Cases of coma occurring in the course of severe illnesses may be readily excluded; it is coma coming suddenly or rapidly which may be confounded with apoplexy. Fagge points out that *pyæmia* may occasionally be accompanied by a sudden coma closely resembling it. More commonly the conditions to be discriminated are coma from injury, poisoning by opium, alcoholic poisoning, uræmia, and epilepsy.

Cases of *injury*, in the absence of history, may present the greatest difficulties, as even with the external evidence of injury, it may remain uncertain whether the patient has fallen as a result of apoplexy, or has injured his brain in consequence of the fall. Even after death the problem may be insoluble. The position of a scalp wound in relation to the weaker side, if paralysis can be recognised, may sometimes help; and the age of the patient, or other circumstances of his bodily health, may render a spontaneous lesion of the brain more or less likely.

Opium-poisoning is generally distinguished by the minutely contracted pupils, the slow pulse, and slow respiration; but it may be closely simulated by apoplexy of the pons Varolii. Evidences of unilateral lesion, such as greater flaccidity or rigidity of limbs on one side, or unequal pupils, are in favour of apoplexy.

The same may be said of *alcoholic poisoning*. The condition is one of profound coma, without any one-sided symptoms. Evidence of alcohol may, of course, be obtained from the breath, or from the stomach by means of emetics or the stomach-pump. But a patient may have drunk freely or sufficiently just before an apoplectic attack, or if the attack has come on gradually he may have taken a glass of spirits as treatment.

Uræmia is probably always accompanied by albuminuria; but the detection of albumen in the urine does not exclude cerebral hæmorrhage: for, in the first place, hæmorrhage occurs often in those who have granular kidneys; and, secondly, hæmorrhage may itself produce albuminuria in those who have healthy kidneys. In uræmia sometimes the coma is less profound, the patients are more easily roused for a time by shouting, to relapse again into coma. There is no paralytic weakness, no vaso-motor disturbance, and no flush of congestion, such as occurs in some cases of apoplexy. Addison used to call attention to the hissing nature of the stertor. General convulsions alternating with the coma, or a previous sudden amaurosis, are in favour of uræmia.

Epilepsy is sometimes followed by coma, which is more like natural sleep than that of apoplexy; the patients are more easily roused, and there are no unilateral symptoms. Early age would be opposed to apoplexy. On the other hand, a gradual onset excludes epilepsy. Occasionally, *hysterical* patients will lie unconscious for long periods, but the cases are generally distinguished by other characteristic symptoms.

It should be noted that other organic diseases of the brain, such as *tumour* and *meningitis*, may rapidly terminate in coma, but the history will generally be sufficient to prevent mistake.

(2) In the diagnosis of the causes of apoplexy, one has to consider the nature of the attack and the associated condition of the patient. It will have been seen that the nature of the attack often gives but little help. Hæmorrhage, embolism, and thrombosis may all produce a sudden or rapid coma. The more severe and prolonged the coma the greater the probability of hæmorrhage; whereas a pronounced hemiplegia occurring without coma, or with very transient unconsciousness, is more likely to be due to embolism. Age is in favour of hæmorrhage, and youth almost excludes it unless an antecedent embolism is possible. The associated conditions of hæmorrhage are albuminuria and other evidences of renal disease, or arterial degeneration, hypertrophied heart, tense and rigid or thickened arteries, and arcus senilis. Senile changes in the arteries may also be recognised in many cases of thrombosis, and syphilitic changes may be suspected from the history. In embolism there is generally a mitral or aortic murmur, or some evidence of dilatation of the left cavities of the heart, which serves as a source of the embolus; or there may be signs of embolism in other parts of the body, such as enlargement or tenderness of the spleen, blood in the urine, the characteristic appearances in the retina, or obstruction of an artery in one of the limbs.

Treatment.—If cerebral embolism can be certainly recognised, the treatment is similar but more simple than that of cerebral hæmorrhage; as the question of depletion cannot be entertained for a moment. Absolute rest, milk diet, ice to the head if there is pain, and gentle laxatives or enemas if the bowels are confined, are the main indications.

INFANTILE HEMIPLEGIA.

This may be congenital, but is more often acquired in the first few years of life. The congenital cases and those in which the symptoms are first noticed within a few months are probably due to difficult labour, at least in many instances; and sometimes the forceps may be indirectly responsible (birth palsies). Acquired cases are no doubt often due to embolic processes, in association with acute illnesses, like scarlatina, measles, and small-pox. Either during such illnesses, or during convalescence from the same, or apparently spontaneously, children are seized with convulsions, and more or less coma, and on recovery are found to be paralysed on one side of the body. The paralysis frequently recovers up to a certain point, when some muscles become rigid and others become the subjects of irregular and involuntary movements (athetosis or post-hemiplegic chorea, *see* p. 276). These are most marked in the arm, which is flexed at the elbow, with the hand dropped, and the fingers and thumb twisting and writhing about. The leg of the same side is stiff, with slight flexion of the knee and talipes equino-varus, the tendo Achillis rigid, and the toes moving about in a purposeless manner. The gait is limping, and the pelvis is tilted to give room for the swing of the stiff leg. As the child grows the affected limbs do not keep pace with the others, and the arm may be found years after to be one and a half or two inches shorter than its fellow, while the hand is narrower and altogether more delicate in form. The legs show similar but less marked differences. The muscles of the affected arm and leg may be less bulky than those of the opposite side, but are never decidedly wasted; and there is no diminution of the electrical reactions or reflexes. Sometimes the muscles are extraordinarily hypertrophied, probably from the constant involuntary contractions. In some cases convulsions occur in the paralysed limbs; in others the patients are dull, stupid, or idiotic; but in others, again, the mental condition is perfectly normal.

The morbid appearances in cases of this kind are not quite uniform. In some there have been found lesions of the convolutions, resulting in considerable loss of substance, and depression on the surface, a condition which has been called *porencephalus.* Presumably in some instances it has arisen from an inflammation of the cortical grey matter (*polio-encephalitis*). In other cases, as in one I recorded ("Guy's Hosp. Rep.," 3rd ser. vol. xxiii. 1878), a lesion of the central parts of the brain is found. But in nearly all cases commencing in infancy and early childhood, and lasting some years, the brain is found to be much smaller on the side of the lesion, the atrophy affecting the crus, pons Varolii, and spinal cord on the same side, while the cerebellum is smaller on the opposite side. Secondary descending degeneration occurs, but is not invariably present: it was absent in my own case. Probably this atrophy is only a secondary change, whether the previous lesion is situate in the centre or on the surface. It would seem that when a lesion of the brain takes place in an infant, it is the undeveloped condition of this organ and of other parts which in some way determines a course of events, different from what happens in adults. Cases of the kind have been recorded as *atrophy*, or *unilateral atrophy*, of the brain, but the atrophy is due, as well as the symptoms, to the primary lesion. The disorder is sometimes spoken of as *infantile spastic hemiplegia*.

Diagnosis.—This form of paralysis, occurring as it does in children, must be distinguished from infantile spinal paralysis (anterior poliomyelitis). The hemiplegic distribution should raise a suspicion, and the mobile spasm or athetosis, the retention of the nutrition of the muscles, of their electrical reactions, and of the reflexes, will show that it is not due to a cornual lesion.

Prognosis as to recovery is very bad, but the patient's life is not threatened, unless from accidents during fits, or from want of nursing and care in the imbecile cases.

Treatment is of little use. Temporary benefit may seem to follow the use of electricity, and convulsions may be diminished by the bromides. But the paralytic condition itself remains as a lifelong deformity and discomfort. The hindrance to gait was so great in one case that Mr. Davies-Colley amputated the foot, with relief.

MENINGEAL HÆMORRHAGE.

The symptoms of meningeal hæmorrhage are variable and scarcely distinctive. Coma is the most marked feature in these cases; and it may come on suddenly or gradually. It is sometimes preceded by such indications as headache, giddiness, or vomiting. Convulsions occur occasionally, and may be local, unilateral, or general. Paralysis and rigidity are not necessarily marked. Sometimes there is mental disturbance—either excitement, delirium, or dulness.

It will be remembered that blood may extend on to the surface from the interior of the brain; and that cerebral hæmorrhage from injury is mostly meningeal in position.

INFANTILE SPASTIC PARAPLEGIA.

A spastic condition of the legs, or of both legs and one arm, or of all four limbs together, occurring in a child is much more often congenital than when the spasm or paralysis is purely hemiplegic. These cases are always of cerebral origin—in many instances due to lesions at birth by difficult labour, forceps, &c., whereby either the motor convolutions are directly injured, or, as shown by autopsies, blood is effused on the surface of the brain. Several names have been employed to designate these cases, such as *cerebral spastic paraplegia*, *congenital spastic paraplegia*, *diplegia spastica* (Gee). Osler calls those cases in which the arms are affected *bilateral spastic hemiplegia*; and those in which the legs alone are involved, *spastic paraplegia*. Gowers says that the arms are never perfectly free. Nothing may be noticed at birth, but walking is very slowly acquired, and the legs are observed to be gradually more stiff; ultimately, the condition is much like that seen in the spastic paraplegia of adults. The limbs are extended and rigid, there is increased knee-jerk, but ankle clonus is not always to be obtained. Sometimes spasm of the adductors is extreme, and the legs are crossed over one another, in spite of which the child manages to walk—crossed-leg progression. The arms are never so rigid as the legs; there may be some stiffness at the elbow-joint, or the fingers are clenched. More often there is jerky movement, or a movement like chorea, or mobile spasm like that of athetosis. I have recorded a case in which violent starting of the rigid limbs is provoked by a loud noise, or by a sharp tap on the head. Convergent strabismus, oscillation of the eyeballs (nystagmus), mental deficiency, or actual idiocy, and late power of walking are also present in many cases.

Treatment.—This is not encouraging. Practically, the patients remain uncured, though some little improvement may be obtained by massage and manipulation, and in extreme deformities by division of tendons, and by mechanical appliances.

HÆMORRHAGE INTO THE PONS VAROLII.

If hæmorrhage takes place into the central region of the pons, there is generally profound coma, with minutely-contracted pupils and complete paralysis of all four limbs. Death is often very rapid, but it may be delayed some hours or three or four days. Convulsions and vomiting are frequent, and sometimes the temperature rises to a great height before death. If a slight hæmorrhage permits of recovery, there will probably be some degree of paralysis of the limbs, with anæsthesia, irregular facial paralysis, paralysis of the tongue and articulation, and dysphagia.

Hæmorrhage into the lateral region causes the forms of paralysis previously described (p. 273), and conjugate deviation, if it occurs, is towards the paralysed side (p. 277). If the posterior or upper surface of the pons is involved, sugar or albumen may appear in the urine, and this secretion may be abnormally abundant (polyuria). The symptoms are, in part, the same as those which result from hæmorrhage into the medulla oblongata, since these two portions of the nervous system are continuous with one another.

CEREBELLAR HÆMORRHAGE.

This is of rare occurrence, and the symptoms present little that is distinctive of the part affected. In some cases there is a close resemblance to cerebral hæmorrhage, the patient having coma with complete resolution of all the limbs, or with paralysis of one or other side. The hemiplegia is opposite to the side of the cerebellum involved, and is regarded as being due to pressure on the pons. Death may occur in a few hours, or days, or the patient may recover from the apoplexy and remain hemiplegic.

Sometimes the symptoms are more obscure, as in the case of a boy aged fifteen, that came under my notice. There was first severe pain in the head; the next day he was sick and constantly throwing himself about in bed; the following day he became lethargic, and he died in the evening. Abercrombie records the case of an old woman who was seized with sudden coma and vomiting, and died after forty hours. A clot was found in the right lobe of the cerebellum.

ENCEPHALITIS.

There is some difference of opinion as to what changes of the brain may properly be included in the term encephalitis, or inflammation of the brain. The white softening which takes place about blood-clots partakes, to a certain extent, of the nature of inflammation, in the destruction of the nerve-fibres and the formation of granule-corpuscles; but as a similar condition results from embolism and the consequent local anæmia of the brain, it is more reasonable to regard it as a degeneration or necrosis of the tissue, arising, in the case of tumours or hæmorrhage, as a result of pressure. Yellow and red softenings are described by some as the same degenerative process, accompanied by more or less extravasation of blood. Yellow softening is, however, often only a condition of œdema with transudation of blood-pigment.

Red softening also, as already stated, may be a result of embolism, when the tension of the cerebral circulation fills the vessels which have been cut off from their connection with the heart; but in other cases *red softening* appears to be a really inflammatory change. The affected part of the brain is swollen, the convolutions are broader, the grey matter of a deep purple colour, the white matter pink or red, and there are numerous ecchymoses. The microscope shows dilated vessels and extravasated white and red corpuscles. This change also may arise in the brain-tissue adjacent to a tumour, or blood-clot, or inflamed membranes. Injury is also a cause of it; but how far it may arise spontaneously, and independently of the above-mentioned local causes, is a matter of doubt; and especially it must be remembered that a condition very similar may be brought about by a vascular new growth.

Induration or Sclerosis.—Cases of *diffuse sclerosis* or induration of the brain occasionally occur, which are probably of chronic inflammatory origin. Wilks has described some cases of the kind ("Guy's Hosp. Rep.," 3rd ser. vol. xxii. 1877). In one the convolutions were flattened and compressed. On section, the white matter was firm, hard, grey in colour, and encroached on the grey matter, pushing the convolutions away from one another. In another there was *meningo-cerebritis*, and the brain substance was tougher than normal. In a third case the membranes were also inflamed; the left hemisphere presented some red softening, but the right was hard, and brick-red in colour, the convolutions were swollen to twice their size, and the cortical was scarcely distinguishable from the medullary matter. Under the microscope the tissue showed an "absence of anything like nerve-structures, and appeared to consist mostly of vessels and a dimly fibrillated substance." In the case of a child, aged eighteen months, recorded by Fagge, the brain was distinctly indurated, the white matter was of a yellowish colour, the grey matter appeared normal, but the pia mater was firmly adherent. The only histological change noted was a slight excess in the cells of the neuroglia. In other cases atrophy of nerve-fibres and increase of the neuroglia are described.

Polio-encephalitis has already been referred to in connection with infantile hemiplegia (p. 289). The brain-substance is also inflamed in wide-spread diseases, *disseminated* or *multiple sclerosis* (*see* p. 251), and *acute disseminated encephalo-myelitis* (*see* p. 220).

Symptoms.—Local encephalitis or softening may be expected to present symptoms in accordance with its position in the brain. Where the changes occur around pre-existing disease, the symptoms may only appear as an extension or aggravation of those due to the primary lesion. The increased headache or relapse of coma, which sometimes occurs after partial recovery from an attack of apoplexy, may be sometimes due to these changes. Cases of more general encephalitis, as Wilks pointed out, have, as a rule, no symptoms of irritation, such as intense pain, delirium, or convulsions; but, on the other hand, the functions of the brain are in abeyance, and the patient becomes drowsy or unconscious, with, perhaps, ill-defined paralysis of one or other side of the body. In the early stages there may be stupidity, inability to speak, indifference to food, loss of sight, hearing, or memory, or tingling and pain in the extremities. But as meningitis was present in some of these cases, we might expect sometimes to have symptoms of irritation, unless the meninges are always affected secondarily.

ABSCESS OF THE BRAIN.

Ætiology and Pathology.—It might be thought that red softening, if of an inflammatory nature, would end in abscess, and in some pyæmic cases patches of red softening have occurred side by side with developed abscesses; but in other cases there is no obvious connection between the two processes. Abscess is rarely, if ever, a primary lesion. It often arises in consequence of suppurative disease of the ear. For instance, otitis occurs during convalescence from scarlatina, the membrana tympani is perforated, and there is a discharge of pus, which may continue for months or years. Ultimately, and sometimes without any apparent cause, the symptoms of cerebral abscess develop. In some such cases, the bone forming the wall of the tympanum is necrosed, the dura mater over it inflames or sloughs, and the pia mater becomes adherent; in others, the bone may be healthy, and the infection seems to have been carried by channels in the bone to the interior of the skull. Even where the membranes are directly inflamed, the abscess may not be in immediate contact with them. The pus from the ear is sometimes fœtid, but not necessarily so. Another cause of abscess is disease of the nasal fossæ or frontal sinuses, and it may follow any other lesion involving the cranial bones, such as direct injury, syphilitic caries or necrosis, or tumour of the bones.

Cerebral abscesses occur in general pyæmia, and another source for them is occasionally seen in inflammatory lesions in the lungs, such, for instance, as phthisis, pneumonia, empyema, and bronchiectasis. It is possible here that particles of thrombus are carried from the lungs into the general circulation, and so to the brain. In some cases of cerebral embolism from infective endocarditis the softened tissue breaks down into a fluid indistinguishable from pus.

The position of the abscess is determined, to a certain extent, by its cause. Thus, if due to otitis, it is mostly in the temporal lobe; or in the cerebellum, if the mastoid cells are especially involved. And disease of the nose may give rise to abscess in the frontal lobes, but the rule is not universal. Generally, there is only a single abscess, but in pyæmia there are often two or more, situated indiscriminately. They are, however, commonly located in the white matter of the hemispheres, or of the cerebellum, and rarely in the grey matter, or at the base of the brain. They vary in size, and may reach two inches in diameter. Recent abscesses have a shreddy wall, older ones have a definite and often thick cyst wall or capsule, composed of fibrillated, if not fibrous, tissue. The pus is mostly greenish in colour, viscid, and acid in reaction; but in long-standing cases it becomes more mucoid still, alkaline, and of a bright green colour. Sometimes, especially when due to bone disease, it may be extremely offensive. The brain-tissue outside the abscess may be softened. Though in many fatal cases the abscess is found intact, it may rupture on the surface, and set up meningitis; or into the lateral ventricles; or it may form a communication, through diseased bone, with the tympanum, and discharge externally (*otorrhœa cerebralis*).

Symptoms.—These are often extremely obscure. The most constant is *pain*, of a continuous dull aching character; or more severe, so that the patient holds his head with his hands, or bores his head into the pillow, or cries out constantly. Exacerbations of the pain occur from time to time. The seat of the pain often, but not always, corresponds to the position of the abscess. Sometimes there is elevation of the temperature, sometimes *rigors*, either occasionally or following with such regularity as to suggest ague, and sometimes profuse perspiration. Convulsions and vomiting may occur. Optic neuritis is much less frequently present than in cases of tumour. Alterations in manner, dulness, listlessness, loss of memory, and emaciation are also sometimes observed. The common seat of the abscess renders localising symptoms on the side of the motor tract or nerve-trunks rather improbable; but there may be ill-defined hemiplegia, or aphasia, or some implication of one or more of the cranial nerves, and in abscess of the cerebellum, some uncertainty of gait.

The duration of the symptoms is very variable; they may last for months, or they may end fatally in a few weeks. Death is often quite rapid, the patient becoming delirious, or quickly drowsy and comatose. Respiration may cease before the pulse, as in the sudden deaths from cerebral tumour.

Diagnosis.—The diagnosis of abscess of the brain is not always easy; its pain may be mistaken for neuralgia, and its rigors for ague.

The most important factor in diagnosis is the presence of a primary cause, and chronic discharge from the ear is the most frequent of these. It must not, however, be too hastily assumed that acute head pains and pyrexia occurring in a patient the subject of chronic otitis are due to cerebral abscess, even though rigors and optic neuritis be present as well. For otitis may produce, besides abscess of the brain, subdural abscess, or meningitis, or suppuration of the mastoid cells with or without thrombosis of the adjacent veins and sinuses. All of these are accompanied by severe head pains and fever; with mastoid abscess there may also be rigors. And it is important to remember, what has now been verified in numerous cases, that in mastoid suppuration there is often double optic neuritis, with an entire absence of meningitis or of abscess, as proved by *post-mortem* examination, and by recovery after simply trephining the mastoid cells. The cause is probably thrombosis of some cerebral sinuses. This warning, however, applies almost more to meningitis than to abscess, since optic neuritis is less frequent in the latter. Suppurative meningitis—the form most likely to be confounded with abscess, since they have a common origin—is more rapid in its course, and is more likely to be accompanied by paralysis and fits; the temperature is more uniformly high, and shivering is absent.

Prognosis.—This is very unfavourable, but not always hopeless.

Treatment.—Where an abscess can be with certainty recognised, and its locality accurately determined, there is a possibility of evacuating the pus by surgical means. Several successful cases, thus treated, have been recorded in recent years.

For exploratory purposes bone may be removed with the trephine or with the gouge, and a fine trocar can then be introduced. In cases arising in connection with diseases of the ear, it is desirable that before trephining, a thorough exploration and antiseptic treatment of the tympanum and mastoid cells should be undertaken, both to exclude the possibility of the symptoms being entirely due to these parts, and also, as Barker suggests, to minimise the risk of meningitis during the longer operation.

Apart from surgical interference, the treatment of abscess of the brain must be purely symptomatic; the relief of pain may be attempted by local anodynes, by ice to the head, and by bromide of potassium, butyl-chloral hydrate, or even morphia, internally; large doses of quinine (5 grains every four hours) may also be given, in the hope of neutralising the septic condition.

MENINGITIS.

In the cerebral as in the spinal meninges, we have to distinguish an inflammation of the dura mater, or *pachymeningitis*, and inflammation of the pia mater, or *leptomeningitis*. It will be convenient to begin with the pia mater, and to take, first of all, the form of leptomeningitis which is associated with tubercular infection.

TUBERCULAR MENINGITIS.

Ætiology.—This disease occurs at all ages, but is generally regarded as more frequent in children than in adults; and it certainly affects males more than females. So far as its causation is concerned, it is constantly associated with tubercle elsewhere in the body. In numerous cases it arises in the course of phthisis, hip-joint disease, caries of the spine, or other tubercular or strumous complaints, and is then sometimes called *secondary*. Other (*primary*) cases, which are especially frequent in children and young people, seem to arise in persons previously quite healthy, or at most after a few weeks' malaise; but even in these instances, after death, it is nearly always the case that some other lesion is present, such as caseating bronchial glands, or miliary tuberculosis of the lungs and other viscera, or a caseous nodule in the brain itself. There may be discharge from the ear, but, if it has any relation to the disease, it is either that it indicates general ill-health, or that it opens a passage for the entrance of tubercle-bacilli.

Morbid Anatomy.—The characteristic appearances are seen in the pia mater, and consist of the effusion of lymph, and the presence of tubercles. The lymph, which is gelatinous and translucent, or opaque and grey, or greyish yellow, but rarely or never distinctly purulent, is contained in the meshes of the pia mater, especially at the base of the brain, over the optic chiasma, the diamond-shaped space behind it, and the adjacent crura and pons. From this central point it commonly extends into the Sylvian fissure on each side, along the course of the middle cerebral artery, where it may be very abundant. The surface of the hemispheres is commonly free from lymph, or at the most a little dull, or sticky, so that tubercular meningitis is often called a *basal* meningitis; but it is common to find a small patch of lymph at the top of the cerebellum, at the anterior part. With the lymph are commonly mixed tubercles, varying from mere points up to the size of millet seeds, generally grey and opaque, occasionally commencing to caseate. The tubercles are especially abundant on the branches of the Sylvian arteries, and in the membranes between them. By separating the arteries with their branches from the brain, and floating them in water, the tubercles may be seen as minute thickenings upon the capillary branches. Under the microscope the smaller tubercles present aggregations of lymphoid corpuscles in the perivascular sheath; the larger tubercles may present all the characteristic features, giant cells, and bacilli. The relation of the inflammatory lymph to the tubercles is very variable. There may be abundant lymph in the characteristic situations, with few, if [illegible] there may be a good number of [illegible] the lymph. Occasionally, cases are fatal [illegible] indistinguishable from those of tubercular menin-[illegible] are found on the surface, and no evidence [illegible] The ventricles of the brain are commonly dis-[illegible] the name *acute hydrocephalus*), the [illegible] flattened against the skull, the fornix and septum [illegible] softened, and the ependyma of the ventricles [illegible].

[illegible] usually affected, but the spinal [illegible] tubercles, and the lymph in [illegible] the cervical region of the spinal [illegible] a general tuberculosis is not [illegible] thickly studded, and there may be [illegible] spleen, and kidneys. With regard to both [illegible] cases the meninges are, no doubt, infected [illegible] from a preceding tubercular deposit in lung, [illegible] gland, brain, or elsewhere; or possibly, in some [illegible].

Symptoms. [illegible] will first be described as they occur in the [illegible] in children, and the differences in secondary [illegible] mentioned. There is often a prodromal [illegible] the child is out of health, restless, loses [illegible] may be occasionally sick, and has consti-[illegible] begins more definitely by headache, or [illegible] convulsion. The headache is severe and [illegible] from time to time; the child puts [illegible] and may be often crying, "Oh, my head!" [illegible] moaning, or occasionally uttering a sudden [illegible] there is a moderate degree of fever, [illegible] sensibility to light and sound, so that the [illegible] desires to be left alone in bed; resents [illegible] often curls itself up in bed away from [illegible] vomiting does not generally last long. [illegible] a fit, this is not often repeated. [illegible] and there may be diplopia quite

[illegible] with severe headache, there may be [illegible] becomes drowsy. The head is [illegible] neck is stiff; the abdomen becomes [illegible] the outline of the muscles being [illegible] margins of the ribs and the iliac [illegible] the terms *carinated* and *boat-*[illegible] pulse may be slow, and is often [illegible] sighing and irregular; the [illegible] or oscillates between 101° [illegible] paralysis is seen in the flushing of the face, and the production of patches of redness wherever pressure is for a time applied. When the finger is drawn sharply across the skin of the forehead or abdomen a broad red line quickly appears, and may persist five minutes or more. This condition which is not peculiar to, but only more marked in, meningitis, is called *tache cérébrale*, *tache méningitique*, or cerebral streak. Even as early as this changes may often be observed in the optic disc, which at first becomes highly vascular, and then shows definite optic neuritis. Tubercles are seen in the choroid in a small proportion of cases. Food is taken badly, and the bowels are constipated.

From this point the case may steadily go on to a fatal termination, without any fresh symptom. The drowsiness increases to coma, optic neuritis is more pronounced, the abdomen becomes more and more hollowed, the pulse more irregular, feebler, and generally quicker, the respiration may take on the character of Cheyne-Stokes' breathing, and the temperature may fall more or less rapidly, or just before death go up quickly to 106° or 107°. Mucus accumulates in the bronchial tubes, and with failing pulse death takes place. But often the last two or three days are marked by local symptoms. An arm or leg, or an arm with the leg of the same side, becomes either rigid or paralysed; or there is slight facial paralysis, or squinting, or ptosis. The pupils are frequently unequal, and one or both may be insensitive to light. Frequently this stage is marked by convulsions, and these may recur several times before death. With the development of these symptoms coma becomes more profound, and death takes place as above shown, or the patient is asphyxiated in a convulsion.

The duration of the illness varies between ten days and three weeks from the beginning of the pronounced symptoms; but occasionally it may be four, five, or six weeks. The above course of the disease has been divided into three stages—a stage of *irritation*, one of *compression*, and the last, a *paralytic stage*. But it is not always easy to distinguish between them, and in some cases the more typical symptoms may be very little marked, coma alone being prominent.

In *secondary* tubercular meningitis the symptoms are often much more rapidly developed, and more insidious. They may, of course, be masked by those of the disease already existing. The patient may, with very little warning, become delirious, or have paralysis of a limb or of the face, or have a fit, quickly becoming comatose, and dying within a few days.

Diagnosis.—This is sometimes comparatively easy; at others difficult or impossible until quite late in the illness. The fact of meningitis is to be suspected when decided head symptoms are accompanied by fever; but with headache alone the diagnosis can rarely be conclusive. In young children, for instance, *otitis* may cause severe headache and moaning, vomiting, photophobia, and the desire to lie undisturbed. A careful examination may show that the pain is more or less localised, or that the ear or mastoid process is tender. *Enteric fever* may for some days simulate meningitis in the headache, drowsiness, and fever; but in enteric, headache rarely persists after the tenth day, and generally by that time the characteristic loose yellow stools, or the rose spots on a full abdomen or the Widal serum-test will decide the diagnosis, which will be confirmed, as the case goes on, by the entire absence of convulsion, rigidity, or paralysis. The mistake is sometimes made in the other direction, cases of meningitis, without any prominent headache, but with flushed face, delirium, and pyrexia, being regarded as enteric fever. The most useful indications here are the irregular pulse, sighing or irregular respiration, rigidity of muscle, or paralysis, convulsions, and optic neuritis. But the last occurs also in enteric fever, although rarely. Tubercle of the choroid is by no means common, and with few exceptions, the diagnosis has to be made without it. In young children, decided cerebral symptoms accompany other acute illnesses, as, for instance, *pneumonia* and *broncho-pneumonia;* the child is often drowsy, with retracted head, and towards the end convulsions may occur. These symptoms would be quite explained by the detection of localised dulness with bronchial breathing, but râles all over the chest might be the indication of a general tuberculosis. Another condition that may simulate it to a certain extent is the *exhaustion* following upon malnutrition, bad feeding, or severe diarrhœa in quite young infants. The child is drowsy or comatose, with pale face, sunken eyes, dilated irregular pupils, and irregular, sighing respiration. It was formerly called *hydrocephaloid disease* or *spurious hydrocephalus.* It is distinguished from meningitis by the history, the absence of fever and local paralyses, the depressed fontanelle, and the speedy improvement under restorative and supporting treatment.

The distinction of tubercular from suppurative meningitis rests largely upon (1) the absence of local cause for a suppurative meningitis, such as cranial injury, or otitis; (2) the previous existence of tubercular lesions, such as phthisis; (3) the paralyses of cranial nerves, indicating that the meningitis is situate at the *base* rather than over the vertex; (4) the duration, which is commonly very much shorter in suppurative meningitis, even two or three days only. It is much more difficult to distinguish between tubercular meningitis and the non-tubercular posterior basal meningitis of infants (*see* p. 304); that is, the localisation in some cases of tubercular meningitis may be precisely the same as is common in the other class of cases.

Prognosis.—Undoubtedly, tubercular meningitis is in nearly all cases fatal. But a certain number of patients who present the evidences of spontaneous, and possibly tubercular meningitis, get well, and though the tubercular nature of the meningitis may be incapable of proof, it is generally believed that some such cases have been of this kind. The recovery is often slow: speech, vision, and the power of walking remain imperfect for weeks or months, thus showing conclusively that there has been a serious interference with the cerebral functions. The prognosis, therefore, though in the main unfavourable, need not be absolutely hopeless in primary cases; but we may regard cases supervening upon phthisis, hip disease, or other well-marked tubercular lesion, as much more certainly fatal.

Treatment.—With the doubt thrown upon the curability of the disease, the subject of treatment seems reduced to small limits. Cold should be applied to the head by means of an ice-bag, the bowels should be opened, and milk must be given in small quantities frequently. Blisters to the back of the neck, iodoform ointment to the scalp, and other local irritants are of more than doubtful value. Of internal remedies, iodide of potassium is often given in doses of 3 or 5 grains to children, and the bromide in similar or larger doses may help to allay the pain in the head.

SUPPURATIVE MENINGITIS.

Ætiology.—In its causation it presents many resemblances to abscess of the brain: thus, it may follow injuries to the head, or be set up by extension of inflammation in neighbouring parts, such as otitis media, suppuration of the mastoid cells, disease of the nasal cavities, syphilitic caries or necrosis of the skull, suppurative phlebitis, or abscess of the brain. It occurs also as a complication in some general diseases of an acute, febrile, or infective nature—pyæmia, septicæmia, malignant endocarditis, enteric fever, small-pox, scarlet fever, and sometimes pneumonia. Meningitis occurs also as a tertiary syphilitic lesion, but is then more often subacute or chronic. But cases not infrequently occur in which, with the most careful search, no primary lesion can be found, and these must therefore be regarded as idiopathic. Some writers would regard these as sporadic instances of the epidemic cerebro-spinal meningitis already described (p. 82).

Pathology.—The inflammation chiefly affects the pia mater and arachnoid (*leptomeningitis*), the purulent effusion lying either in the arachnoid cavity (subdural), or much more frequently in the meshes of the pia mater itself. When it has spread from a diseased bone of the skull, the dura mater itself may show localised inflammation, but the extension of the disease over the brain is by means of the other membranes. Commonly, the convex surface of the brain presents a more or less extensive layer of bright yellow or green pus, which may be on both sides, or confined to one side, the side of the lesion in secondary cases. The pus frequently follows the course of the larger vessels, and dips down with the pia mater into the sulci. Though mostly affecting the upper surface of the hemispheres (meningitis of the convexity), it may extend to the base, or the pus may find its way, perhaps by gravitation, into the spinal canal. The brain-tissue beneath it is commonly softened, and may present ecchymoses or minute abscesses.

Symptoms.—While there is a general resemblance to the symptoms of tubercular meningitis, the course of acute meningitis is usually much more rapid, and there is much diversity as to the prominence of particular symptoms. Where meningitis supervenes upon other acute illnesses, its features may be more or less masked. In idiopathic cases, or cases caused by chronic inflammatory lesions, like otitis, the symptoms often commence acutely with chill, or rigor, and acute pain in the head. This is generally very severe and constant, but aggravated from time to time. With this the patient becomes feverish, shuns light and noises, and may lie curled up in bed, resenting interference, as in the tubercular cases. Vomiting often occurs at the commencement. There may be rigidity of the muscles at the back of the neck, and the head is drawn back. The pupils are often contracted. Drowsiness and delirium soon supervene, and convulsions and paralysis supply further evidence of the interference with the cerebral functions. A convulsion may, indeed, be among the first symptoms; in the later stages the fits may be often repeated, and they are generally bilateral. The paralysis is very variable, corresponding to the situation of the effusion; from its frequent occurrence at the vertex it less often affects the cranial nerves than does tubercular meningitis, though there may be squint; but an arm or leg is often paralysed, or there may be complete hemiplegia. Sometimes there is rigidity of the paralysed limbs, or of their fellows, with increasing coma. The pupils become dilated, and the ophthalmoscope generally reveals optic neuritis, which may develop rapidly under observation. The temperature is high, varying from 102° to 104°, the pulse is mostly rapid, respiration is sighing, irregular, or of Cheyne-Stokes' type, *tache cérébrale* may be well marked, and in some cases the abdomen is retracted. Finally, the evacuations are passed involuntarily, the breathing and circulation fail, mucus accumulates in the chest, and death terminates the scene. The course of the disease is rapid; it is often fatal within two or three days of the first symptom; exceptionally the illness may last longer, as in a patient under my care, who died on the twentieth day.

Diagnosis.—This presents the same difficulties as in tubercular meningitis, but the course being much more rapid, it is less often the stage of headache than the stage of coma, or delirium, that may be mistaken. Sometimes a diagnosis has to be made when severe headache, and a quickly following coma, are the only important features of the case. Where a primary source for the meningitis, such as otitis, exists, one may be easily led to a right opinion; in the absence of this, one must look for fever, or any indication of paralysis or rigidity of a limb. As compared with ordinary *apoplexy*, the hemiplegia of meningitis is often much less complete; it may, however, be a typical hemiplegia, so far as its distribution is concerned, from the meningitis involving the cortical motor area. Meningitis has to be recognised as one of the results of *chronic otitis*, and the difficulties in determining its presence in that disease have been already pointed out, with the help which may be derived from the use of the ophthalmoscope (p. 296). The symptoms of meningitis may also be confounded with those of *abscess*, and all the more, as either may occur from disease of the ear, or of the cranial bones. The complete clinical course of fever, headache, delirium, coma, and paralysis or rigidity, all within three or four days, is in favour of meningitis; in abscess there is more likely to be severe headache for some days before the coma—the temperature is either lower, or oscillating, with chills, rigors, or sweating. The diagnosis from *tubercular meningitis* has been already discussed (*see* p. 300). By relying too much upon the mental condition of meningitis (and abscess) in young women, an unjustified suspicion of *hysteria* may for a time be entertained.

Prognosis.—The majority of cases of suppurative meningitis are fatal. What proportion, or if any at all, recover, is still much debated, because *post-mortem* evidence is not forthcoming to prove conclusively the actual occurrence of meningitis. A child under my care recovered after prolonged semi-coma, retraction of the head, rigidity of the limbs, and blindness, sight and the power of walking only slowly returning during the next few months. In more acute cases of meningitis recovery sometimes takes place, but the prognosis must be unfavourable in proportion to the rapidity and severity of the symptoms. Possibly, the stage of suppuration is not reached in patients who survive.

Treatment.—The application of cold to the head, by means of ice-bags, is the chief local means; with intense pain, leeches might be applied to the temple. The use of blisters, or mercurial ointment, is of doubtful value. Internally, bromide of potassium may be used to relieve pain, and mercurials and iodide of potassium may be given in the hope of influencing the morbid process. Where a syphilitic origin is certain, these should, of course, be pushed to full doses. The bowels should be kept active, and fluid nourishment given in small doses frequently.

POSTERIOR BASAL MENINGITIS OF INFANTS.

Some cases of simple meningitis occur among children, characterised by convulsions, rigidity, opisthotonus, and especially by retraction of the head (cervical opisthotonus). Gee and Barlow recorded twenty-five such cases, and of fourteen, which they watched to the end, only three recovered. The *post-mortem* examination, in six of the fatal cases, showed a basal meningitis, without any tubercle of the brain or the membranes, and variable effusion into the ventricles. In two cases there was also spinal meningitis; in another case it was absent.

Some of these cases certainly recover. Dr. Still has recently found in cases of this kind a diplococcus, smaller than the pneumococcus, and, as he believes, identical with the diplococcus intracellularis of Weichselbaum. It was present in the membranes, or in the ventricular fluid, but not in the blood.

CEREBRAL PACHYMENINGITIS.

The dura mater becomes inflamed on its outer surface (*pachymeningitis externa*) in consequence of injuries, or the extension of inflammation from diseased bone, otitis, or any of the causes already mentioned as leading to suppurative meningitis. The inner surface of the dura mater is also inflamed in many cases of suppurative meningitis (*pachymeningitis interna purulenta*). The symptoms in these cases, due to the inflamed dura mater, are not distinguishable from those referable to the other membranes.

A third affection of the dura mater is that known as *pachymeningitis interna hæmorrhagica*, or *hæmatoma of the dura mater*. Virchow's view that this is primarily an inflammation with subsequent bleeding into the newly-formed tissue is now generally held, though it has been also thought that it might originate as a hæmorrhage with organisation into fibrous tissue, in which, again, new vessels and fresh hæmorrhages take place.

Ætiology.—Hæmatoma has been found most commonly in association with chronic insanity and chronic alcoholism; it also occurs in old people apart from these conditions, and in some local affections of the brain, such as apoplexy, softening, and tumour. A condition of general or local atrophy of the brain, with degeneration of the arteries, is common to all these conditions. The disease is much more common in elderly people, and more frequent in males than in females. A primary hæmatoma may arise from injuries to the skull, and probably from other conditions likely to produce hæmorrhage, such as chronic affections of the heart and lungs, and diseases of the blood.

Morbid Anatomy.—The inner surface of the dura mater is covered with one or more layers of membrane, soft and friable when recent, tougher and more fibrous when old; in colour brownish-red, brown, yellow, or even white, and often presenting punctiform ecchymoses; while between the layers may be considerable quantities of blood-clot more or less altered by age, or collections of serum containing cholesterin crystals. The deposits are usually situate over the parietal region, near the middle line, and are bilateral in about half the cases. They may be thick enough to depress the surface of the brain. Whether the first deposit is inflammatory or hæmorrhagic, it is clear that there is a new formation of tissue, with newly-formed capillaries, and that subsequent bleedings take place from these vessels.

The **Symptoms** are very variable. Often the condition has been found *post mortem* without any symptoms which would be explained by it; sometimes, on the other hand, a fatal apoplexy is the result of a large hæmorrhage between the membranous layers, which compresses the brain. Such an illness probably cannot be diagnosed from other forms of cerebral hæmorrhage. But the attack may be less severe, and recovery take place; or there are fresh seizures at different intervals. The symptoms are generally headache, giddiness, somnolence gradually increasing to coma, and twitchings or convulsions in the limbs and face of one side, followed by muscular weakness or definite paralysis. The pulse is often slow or irregular, the pupils are contracted, and there is some degree of fever. In the intervals the patient may return to his former condition, or present some impairment of the cerebral functions, such as diminished intelligence and memory, drowsiness, weakness of limbs, and headache.

Diagnosis.—Huguenin mentions, besides the predisposing conditions, the evidence of sudden and increasing compression, the symptoms showing that the convexity, and especially the cortical motor area, is affected (unilateral convulsions, followed by paresis, absence of oculo-motor paralysis), spreading of the disease from one side to the other, and recovery after apparently severe illness. Cerebral symptoms in the interval, and one or more previous attacks, strengthen the diagnosis.

The **Treatment** should be similar to that of apoplexy.

THROMBOSIS OF THE CEREBRAL SINUSES.

The blood coagulates in the cerebral sinuses either as a result of general ill-health, or in consequence of local lesions leading to phlebitis.

The former causes mostly an adhesive thrombosis, the sinus being obstructed by laminated clot, without any general infection of the system. It occurs most often in infants, especially those suffering from marasmus or chronic diarrhœa. It mostly affects the longitudinal sinus. Coma, stiffness of the back, neck, or limbs, strabismus, nystagmus, and paralysis or spasms of the face, are said to occur. Distension of the veins over the forehead and temple, and epistaxis, have been described as resulting from thrombosis of the longitudinal sinus, and œdema of the mastoid process when the lateral sinus is affected; but the constancy of the symptoms is doubtful.

In adults the chlorosis and anæmia of young girls is an occasional cause: but phthisis, cancer, and wasting diseases more commonly. The symptoms are similar—namely, vomiting, headache, drowsiness, convulsions, delirium and coma, with nystagmus, strabismus, sometimes optic neuritis, and twitchings or weakness in the extremities. The result depends largely upon the primary ill-health, to which treatment must be directed.

Thrombosis from *phlebitis* is mainly caused by extension from disease of the ear, and is a fertile source of danger. The lateral or petrosal sinus is most often affected; and it has already been stated that the optic neuritis frequently accompanying the spreading forms of otitis is probably due to this. Thrombosis of the lateral sinus may extend into the jugular vein, and produce a hard swelling, with more or less tenderness, or even redness, œdema, and finally suppuration, in the upper part or whole vertical extent of the neck. Commonly, in such cases septic particles are conveyed into the right heart, and thence into the lungs, so that a fatal pyæmia is the result.

Treatment.—In thrombosis dependent on general ill-health, this condition must be treated. If local lesions are the cause, they must be dealt with surgically. The spread of an infective thrombus down the jugular vein can be prevented by tying the vein below the clot and cleaning out its contents, as well as those of the lateral sinus, if necessary.

TUMOURS OF THE BRAIN.

Tumours are much more frequent in the brain than in the spinal cord; for the most part the same kinds are found in both organs.

The most frequent form of tumour of the brain is *sarcoma*. It often grows from the membranes, or commences in the skull and passes through the membranes to the brain. It may be a primary tumour, but is often secondary to growths in other parts of the body, and is then frequently multiple.

The next most frequent form of tumour is *glioma*. This consists of an overgrowth of the connective tissue, or neuroglia, of the brain. It may reach a diameter of two or three inches, is ovoid or globular in shape, and infiltrates the tissue of the brain in such a way that the divisions into grey and white matter are often perfectly apparent in their normal relations, though perhaps separated from one another, or, as it were, enlarged. The tumour is thus never encapsuled, but gradually shades off into the surrounding normal tissue. Under the microscope it consists of small cells, and of fine fibres forming a looser or denser network. In some cases it can be seen that the fibres are delicate processes connected with the cells. Sometimes the cells are of larger size, or the fibres less abundant, and thus they come more and more to resemble a sarcomatous tumour. The term *glio-sarcoma* is often used for these forms. Gliomata are very liable to hæmorrhage in their interior from rupture of vessels.

Tubercle occurs in two forms in connection with brain: first, in the form of minute tubercles in the membranes, in association with meningitis; secondly, as large masses in the cerebral substance, ranging from one-third of an inch to two or three inches in diameter. These are globular in shape, consist of bright yellow caseous material, bounded by an outer narrow zone of a pinkish-grey colour, while the adjacent brain-tissue is either normal, or slightly softened, with granule-corpuscles visible under the microscope. The tubercular masses can be easily detached from their bed. The grey edge of the tumour consists of cells resembling those of grey tubercle; the centre of the caseous portion may be softened into fluid or may be calcareous. These tubercular tumours are found in all parts of the brain, often in the cerebellum. They are sometimes multiple, and not infrequently accompany tubercular meningitis. They come in order of frequency next to glioma, and in children are proportionately more frequent.

Carcinoma is not very common: it is nearly always secondary. *Myxoma* and *fibroma* are rare.

Cysts occasionally occur, sometimes obviously as a result of a soft sarcomatous tumour breaking down in the centre; at others, consisting of a simple membrane containing serous fluid, without any trace of a previous new growth. These have been found in the cerebellum.

Psammoma is a peculiar tumour, which grows from the membranes, and consists of fibrous tissue, with embedded particles of calcareous matter. *Cholesteatoma*, or pearl tumour, is another rare variety, which grows from the pia mater of the base of the brain; it is hard, shining like mother-of-pearl, non-vascular, and composed of horny epidermic cells arranged in concentric layers, and enclosed in a fibrous capsule. *Lipoma*, *angioma*, and *melanoma* have also been observed.

Syphilitic Gumma.—This form of tumour grows commonly on the surface of the brain, and at first in the pia mater; it subsequently invades the substance of the brain, and contracts adhesions to the dura mater, so that the brain-substance and the two membranes are matted together. The tumour is irregular in shape, pinkish-red on section in the outer parts, yellowish and caseous in the centre. Sometimes syphilis causes a localised and yet diffuse meningitis, without the formation of a definite gummatous tumour; and this is especially liable to occur at the base of the brain.

Parasites.—These rarely reach the cerebrum. A *hydatid* cyst (echinococcus) may grow in the hemisphere, and produce all the symptoms of tumour. The *cysticercus telæ cellulosæ* has been seen in the membranes and in the ventricles.

Ætiology.—This varies to a certain extent with the nature of the tumour. Some, as already stated, are secondary to new growths elsewhere. Tubercular tumours often occur in association with caseous changes in the bronchial or other glands. Syphilitic lesions are mostly tertiary, and never in connection with the congenital form of the disease.

Males are more often the subjects of cerebral tumour than females; and the most striking fact about the age is that tubercular tumours are much more common in children. Fagge noted that in tumours of the hemispheres the patients were mostly above thirty, while in tumours of the base they were mostly below that age. In a good many instances the symptoms have been attributed to a fall or a blow upon the head.

Local Effects.—In the immediate neighbourhood of a tumour the brain-substance is frequently affected, either by red or white softening, or by the yellow discoloration, which has been called yellow softening. If the tumour is large, the corresponding hemisphere of the brain is increased in size, and the convolutions are flattened. Even the cranial bones may be thinned by compression, so that they may be indented by the finger; this condition is known as *craniotabes.* Tumours in the middle lobe of the cerebellum or in the posterior lobes of the brain are frequently accompanied by ventricular distension or hydrocephalus, which is attributed to compression of the veins of Galen by the tumour.

Symptoms.—These are (1) *general*, and (2) *localising* or *focal.* The former are present in the majority of cases and depend probably upon the increased intracranial pressure which any addition to the contents of the skull must cause; the latter are those which vary with the position of the tumour, and will help to show exactly where it is situate.

General Symptoms.—*Headache* is usually present, is often very severe indeed, and may be constant; but sometimes diminishes or disappears for a time. It may be felt all over the head, or it is limited to one region, and may serve as a localising symptom. Thus, there is generally occipital headache in cerebellar tumour, and, in a case recently under my care, a tumour of the right superior temporal convolution was accompanied by intense pain above the right ear. Headache is often absent with tumours of the motor area, or of the corpus callosum.

Vomiting is also a frequent and sometimes early symptom, and often leads to a wrong diagnosis of gastric disease. In its most characteristic form the food is regurgitated, without pain or effort or nausea, immediately it is put into the stomach; but nausea and straining sometimes occur in purely cerebral vomiting.

Optic neuritis, or inflammation of the optic disc, occurs in the large majority of cases of cerebral tumour. It is almost constant in tumours of the corpora quadrigemina, exceedingly common in tumours of the cerebellum and posterior part of the cerebrum, but absent in more than half the cases of tumour of the corpus callosum, pons or medulla. It is more frequently present when the tumour is a glioma or a cyst, than when it is a tubercle (Martin, in *Lancet*, 1897, vol. ii.). It is almost invariably double, but exceptionally one eye has been alone affected, or much more affected than the other: the tumour is more likely to be on the side of the worse eye. After death the nerve has been found to present evidences of inflammation, and there is frequently an effusion of fluid within the sheath around the nerve.

The fact that vision often persists when optic neuritis is even well marked (p. 176) renders it necessary that this lesion should be looked for, and an ophthalmoscopic examination should be made in any case where other symptoms suggest the possibility of cerebral tumour, even though the patient should state that he sees perfectly well.

After optic neuritis has existed some months, a condition of *atrophy* supervenes from contraction of the fibrous tissue of the nerve. Vision is now generally diminished, but is not always lost in proportion to the apparent atrophy; the pupils are generally dilated and sluggish or inactive to light.

Convulsions are not so frequent as the symptoms already mentioned, and they are very irregular in their occurrence in the cases attended by them; thus, there may be only two or three in the whole course of the illness, or they may be very frequent. Sometimes they are general and epileptiform in character; at others, they are limited to one or other region, and may then acquire a more localising value.

Among other general symptoms are mental changes; the patient may be dull, apathetic, forgetful, sleepy, careless, or untidy. Vertigo sometimes occurs. The pulse may be unusually slow. The nutrition may be unaffected, and the patient may even grow fat; more often, especially towards the end, flesh and strength are both lost. Constipation is common in cerebral tumour.

Localising or Focal Symptoms.—These may be absent, as in some tumours growing in the cerebral hemispheres. It is scarcely necessary to enumerate all the symptoms or combinations of symptoms which may occur.

Tumours which compress *nerves* or destroy nerve-centres will cause a cessation of their functions—paralysis in the case of motor nerves, and pain or anæsthesia in the case of sensory nerves (*see* Lesions of Cranial Nerves, p. 183, *et seq.*).

A tumour of the *base* is commonly accompanied by paralysis of the third, fourth, fifth, or sixth nerve; if it extends to the pons, some of the lower nerves may be involved.

Tumours in the lower or outer parts of the hemispheres may implicate the *motor tract* and cause hemiplegia, which generally differs from the hemiplegia due to hæmorrhage or embolism in coming on quite gradually. Spasm, either tonic or clonic, also results from implication of the motor tract by tumours.

Tumours (and other lesions) in the *cortical motor centres* lead to localised convulsions (*monospasm*), or to convulsions beginning always in one part. In these last cases, according to the severity of the temporary disturbance in the part, there may be a twitching, say, of the hand alone, or of the hand and arm, or of all the muscles of the body; and when the convulsions are extensive, consciousness is generally lost. The convulsions are sometimes followed by temporary paralysis in the part in which the spasm begins. *Post-mortem* results have shown that convulsions beginning in the leg, arm, or face, respectively, are frequently due to lesions in the corresponding regions of the motor area—namely, for the leg, the upper part; for the arm, the middle part; and for the face, the lower part of the central convolutions. The name, *Jacksonian epilepsy*, has been given to convulsions of this kind.

Growths in the *cerebellum*, so long as they are confined to one or other hemisphere, may present no symptoms; but if of large size, they may extend forward so as to reach the pons, and involve the seventh nerve, or press downwards upon the pyramids of the medulla oblongata, and thus produce some weakness of the arm and leg on the opposite side. But a tumour in the middle lobe of the cerebellum produces some characteristic symptoms—ataxy, vertigo, oscillation of the eyeballs, retraction of the head, or tetaniform convulsions. Ataxy and vertigo are the most common, and may be among the first symptoms.

The ataxy of cerebellar disease is different from that which forms the characteristic feature of tabes dorsalis or locomotor ataxy. The gait is reeling or staggering, like that of a drunken man; the patient sways from side to side, readily overbalances himself, crosses his legs to regain his equilibrium, and often falls in the attempt.

Experiments have shown a close relation between the parts of the cerebellum injured, and movements of the body or eyes in particular directions; for instance, lesion of the anterior part of the middle lobe causes a tendency to fall forwards; lesion of the posterior part, a tendency to fall backwards; lesion of the middle peduncle causes lateral movements, or movements of rotation; and the eyes are moved upwards, downwards, to the right, or to the left, according to the different points selected for stimulation. But in slowly growing tumours it is not always possible to recognise similar effects, as they are probably counteracted by cerebral influence; but if such occur, they may help to indicate the position of the lesion. Two other occasional effects of cerebellar tumour should be mentioned; one is a peculiar slow tremor of the muscles of the trunk and limbs, which has a certain resemblance to that of disseminated sclerosis; and the other, the globular enlargement of the head, which is due to ventricular distension, and is therefore not peculiar to, but frequently occurs, in cerebellar tumour.

A tumour of the *corpora quadrigemina* causes reeling ataxy of the legs like that just described, associated with double ophthalmoplegia.

Duration.—Intracranial tumours usually last from six months to two years, and occasionally longer. Death takes place sometimes from exhaustion with vomiting, emaciation, and bed-sores; at others, from hæmorrhage into the tumour, if it be a glioma; at others, again, from intercurrent pneumonia. Tubercular tumours may end in a fatal tubercular meningitis. Not infrequently death takes place suddenly and quietly by failure of the respiration, while the heart continues to beat for some little time afterwards. In cases dying slowly the abdomen retracts, *tache cérébrale* may be obtained, and the breathing may acquire the Cheyne-Stokes character; the picture closely resembles that of meningitis.

Diagnosis.—The clinical symptoms above given will mostly serve to distinguish tumours of the brain. If headache and vomiting suggest gastric disease, the ophthalmoscope may prevent a serious error, or a careful examination may reveal uncertain gait or some slight paralysis. Double optic neuritis alone cannot now be regarded as conclusive evidence of a cerebral lesion, much less of tumour, as it may occur in anæmia, in meningitis, and in connection with disease of the ear; but it is of great value in association with other symptoms, and at least it excludes hæmorrhage and embolism, in which it very rarely occurs. Optic neuritis of one eye generally means disease in the corresponding orbit. *Chronic meningitis* may be very difficult to distinguish from tumour, but *acute meningitis* is too rapid in its course, and is generally accompanied by fever; the long history will serve to distinguish the case of tumour in its last stages. *Renal disease* may simulate cerebral tumour in the headache, vomiting, and affection of sight; and even the ophthalmoscope may not at once clear up the case, since the optic disc is inflamed in albuminuric retinitis; and, on the other hand, optic neuritis from cerebral tumour is sometimes accompanied by retinal changes (brilliant white spots) precisely like those common in albuminuric retinitis. Albumen in the urine of course speaks for renal disease, but does not exclude a co-existing brain tumour; a constant localised pain, any local nerve-symptoms, and pronounced double optic neuritis, without further retinal changes, are in favour of cerebral tumour. As to the nature of the growth, the frequency of tubercular tumours in children should be remembered, and the liability of gummata to occur on the surface. Cases with irregular paralysis of many cranial nerves are often syphilitic. Hughlings Jackson has said that convulsions beginning unilaterally, associated with double optic neuritis, are generally syphilitic. This is an expression of the fact that gumma may occur on the surface in the cortical area, and, like other causes of intracranial pressure, leads to optic neuritis.

Prognosis.—This is very unfavourable. Only syphilitic cases give any hope of improvement, and they frequently relapse again and again; they may indeed be fatal under the most vigorous treatment. It is probable that tubercles sometimes become arrested in their growth or calcify. Fagge records the case of a boy in whom temporary recovery took place, and a calcareous mass was found in a region corresponding to the symptoms; but he died, nevertheless, with cerebral symptoms. The case of another boy, formerly in Guy's Hospital, is possibly an instance. At the age of seven he had staggering gait, occasional fits, optic neuritis, and an enlarging head. After six or nine months the symptoms progressed no further; and I saw him years later with a large head, unsteady gait, white atrophy of the discs, and vision so defective that he could only read on placing the book within a few inches of his eyes. But such instances are exceedingly rare.

Treatment.—In cases obviously syphilitic, potassium iodide should be given in doses of 10 to 30 grains three times daily, or even more, combined with mercurial inunction, and the internal use of mercury perchloride (60 to 80 minims of the liquor three times daily). Even in cases where a syphilitic origin is less certain, the iodide may be tried, since no other drug can be said to have any influence. Tubercles may presumably be benefited by cod-liver oil, sea-air, and other hygienic measures. Arsenic has been also recommended. Hale White has shown ("Guy's Hospital Reports," 1885–86) that only a small proportion of cerebral tumours are in a position to be reached by surgery; and where one is accessible, there is always the fear that it may extend beyond the area indicated by positive symptoms, and so involve a dangerously large operation. Nevertheless, in a few cases the tumour has been successfully removed.

Besides the direct treatment of the tumour in cases suited for it, we may have to relieve symptoms. We may treat headaches by ice to the head, potassium bromide, or small doses of morphia; and sickness, by effervescing salines, and tincture of iodine in 2 or 3 minim doses. Here, also, the aid of surgery has been invoked, and the skull has been trephined to diminish temporarily, at least, the intracranial tension, and resulting pain or other symptom. In later stages careful nursing becomes necessary.

CHRONIC HYDROCEPHALUS.

By hydrocephalus is meant the accumulation of fluid within the cranial cavity, and mention has been already made of the acute secretion of such fluid in tubercular meningitis, on account of which the disease was formerly known as acute hydrocephalus.

A division has been made into *internal* and *external* hydrocephalus, according as the fluid is contained entirely in the ventricular cavities of the brain, or is formed outside between the brain and the skull, in the subdural space. But much doubt exists as to the real occurrence of the latter class of cases, and their symptoms and general features are not materially different from the certainly more common cases of chronic internal hydrocephalus. It is true that in old age, and from other conditions, the convolutions of the brain diminish in size, the sulci widen, and the space in the skull thus left by the disappearance of brain-substance is filled up by fluid. Similarly, a loss by local shrinking of the brain is replaced by fluid on the surface. But this compensatory secretion has none of the effects of true hydrocephalus.

Hydrocephalus occurs in infants, and much more rarely in adults; the yielding nature of the cranial bones at the former age, and their solidity and resistance at the latter, cause some important differences in the course and symptoms.

INFANTILE HYDROCEPHALUS.

Ætiology.—Infantile hydrocephalus is either congenital, or it is first noticed at a certain interval after birth; but the results are essentially the same. The origin is, as a rule, quite obscure. It has been thought to be caused by an inflammation, started perhaps in some cases by blows or other injuries. When arising *in utero*, it has been attributed to blows or falls suffered by the mother. It has been observed to occur in several children of the same family. At all ages the growth of tumours in the cerebrum or cerebellum, so as to press on the veins of Galen, will cause ventricular distension. In young children the bones yield, and the head presents in a moderate degree the characters to be presently described.

Anatomy.—In the obviously internal form of hydrocephalus, the ventricles of the brain contain an excess of fluid, sometimes amounting to a quart or more. It has the characters of cerebrospinal fluid—that is, it has a specific gravity of 1006–1009, contains a small quantity of chloride of sodium, only a trace of albumen, and sometimes urea or cholesterin. The liquid may occupy all the ventricles, or all except the fourth, or the two lateral ventricles alone. By its increasing quantity the substance of the brain is enormously distended, the convolutions are flattened, sometimes reduced to a few lines in thickness, and the basal ganglia are correspondingly thinned out. The aqueductus Sylvii may be distended to the size of the finger when the fluid is in the fourth ventricle; it is often closed when the fourth ventricle is not dilated. In extreme cases the distinction between grey and white matter is lost in the parts exposed to most pressure; the ependyma is often thickened, and contains amyloid bodies, while its surface is covered with fine granulations.

Hydrocephalus is sometimes associated with other lesions of the central nervous system—for instance, spina bifida or syringomyelia.

Symptoms.—The most obvious, and it may be for a time the only, symptom of the disease is the condition of the child's head which results from it. The pressure on the brain is transmitted to the skull, and as this expands outward the head becomes enlarged. The enlargement is often extreme. In congenital cases it may form a serious obstruction to delivery; in others it appears in the first few months of life, and the circumference may amount to twenty-four or even thirty-two inches, instead of sixteen or eighteen inches, which are the usual measurements up to the age of one year. The head is at the same time globular, and the skull projects over the face and neck almost uniformly all round. The face looks small and shrunken in comparison, and has a distressed, anxious, or senile expression in some cases. The distension from within drives the orbital plates downwards, and the eyeballs are turned down so that the lower part of the iris and of the cornea is lost under the lower eyelid, and the upper part of the sclerotic is exposed. The increased size of the head is due to a separation of the cranial bones from one another, so that the fontanelles are much enlarged and the sutures widened. In these spaces fluctuation can sometimes be felt. In cases of long standing it is found that ossification has gone on at the margins of the bones, advancing into the sutures, so that ultimately, if the patient lives, by this means, and by the formation, from independent centres in the membrane, of fresh plates of bone (*ossa triquetra*, Wormian bones), the deficiencies of the skull may be completely filled in. In the early stages, however, the bones are thin, wanting in diploë, and transparent. The skin of the scalp is tightly stretched, excessively thin, and large blue veins ramify over the surface. The hair is generally scanty. In some cases, where the fluid is not very abundant, the bones may yield sufficiently to obviate any considerable pressure upon the brain-substance. The symptoms may not then go much beyond the enlargement of the head; at most there is some general weakness and loss of flesh, from which after a time the child recovers. But in most cases there are other symptoms. The muscular power is deficient; especially, the large head cannot be held upright, and falls from side to side, or has to be supported by the hands when the child sits up in bed. The child cannot walk, or acquires the art only after a long time in moderate cases. Vision is often defective or lost; and in extreme instances there is atrophy of the optic nerves, which has been preceded in some cases, it appears, by optic neuritis. The other senses may be, to a certain extent, impaired. The intellectual functions are often defective. The child slowly learns to talk, continues childish out of proportion to its growth, and is fretful, irritable, or vicious in temper. Nystagmus, rigidity and spasms of the weakened limbs, convulsions, and vomiting occur often in severe cases. Many of these patients die young, relapsing into a condition of apathy or semi-coma, lying in bed with eyes closed or twitching, with rigid limbs, and incontinence of urine and fæces, constantly moaning or whining, and refusing food or else eating voraciously.

Finally, convulsions, or coma, or some intercurrent disease, such as bronchitis, pneumonia, or measles, may end the scene.

In some cases the fluid has escaped by rupture of the integuments or by bursting through the nose or eyes.

The duration is variable. In the mildest cases recovery may take place, or rather the disease is arrested; other patients live to four, five, or six years, or more. I have recorded a case ("Clin. Trans." 1897) where a lad reached the age of sixteen with perfect mental development and physical capacity; and died then with rapid cerebro-spinal symptoms, the ventricles containing thirty ounces of fluid. A few cases have lived to sixty or seventy.

Diagnosis.—Confusion is most likely to take place between this and rickets. The rickety head tends to be cubical in form rather than spherical, the vertex being flattened; the downward displacement of the eyeballs is absent; the limbs may be feeble, but the mental powers are not generally deficient; and the other evidences of rickets—beaded ribs, thickened wrists, sweating of the head, and general tenderness—are present at one time or another.

Treatment.—This is not very promising. Perchloride of mercury and iodide of potassium have been given with no appreciable effect. Mechanical treatment seems to give the best, though not always much, result. This may be in the form of pressure, or the removal of fluid, or both combined.

Pressure is best effected by narrow strips of adhesive plaster carefully applied as follows:—One is carried from the mastoid to the outer angle of the orbit on the opposite side; one from the occiput to the root of the nose; others are carried similarly from the base of the skull between these points across the vertex to points low down on the opposite side; and then a long strip is carried two or three times round the circumference of the head, so as to fix the whole firmly. After ten or fourteen days fresh strapping may be applied. Elastic bands have been used, but they must be employed with caution, from their tendency to produce sloughing by too continuous pressure.

If it be decided to try the effects of puncture, the trocar should be inserted at the outer angle of the anterior fontanelle. Not more than one or two ounces should be removed, and the head should then be strapped. If no harm results a similar quantity may be again withdrawn after two or three weeks. A certain risk attaches to this operation: namely, the occurrence of convulsions, of acute meningitis, or of suppuration within the ventricles.

HYDROCEPHALUS IN ADULTS.

Ventricular distension in adults arises from (1) mechanical interference with the circulation of the brain; (2) meningitis or meningo-ependymitis.

1. *Interference with the Cerebral Circulation.*—This is not peculiar to adults, but the larger proportion of cases probably occurs after the period of life at which any very great expansion of the head can take place. The head nevertheless becomes globular and somewhat enlarged. This is often seen in cases of cerebellar tubercle, or other tumour situate there or in the posterior lobes of the brain, whereby the veins of Galen or the straight sinus may be compressed. The return of blood is thus delayed, and effusion into the ventricles is the consequence. It is not generally possible to distinguish the symptoms due to the hydrocephalus from those of the tumour which causes it.

2. *Meningitis and Meningo-Ependymitis.*—Acute meningitis is rarely a cause of hydrocephalus; sometimes the latter is a sequel of epidemic cerebro-spinal meningitis. But some of the cases of chronic hydrocephalus in adults, which are not the result of

the growth of tumour, show after death evidences of an inflammatory origin, such as thickening and granular condition of the ependyma, thickening, opacity, or matting together of the membranes at the base of the brain, adhesion of the membranes to the brain, &c. The fluid reaches a much smaller amount than in the infantile cases; the convolutions are more or less flattened; the bones are thin, and wanting in diploë. Sometimes the sutures have opened again.

Symptoms.—These are not, as a rule, distinguishable from those of some chronic disease of the brain, such as tumour, and chiefly because the enlargement of the head, due to the expansion of the bones of the skull, is absent. Much variety has been observed in different cases. The symptoms that have been noticed are pains in the head, numbness in the feet and legs, weakness or paralysis of the limbs, sometimes even hemiplegia though the lesion is bilateral, blindness with optic neuritis or atrophy, delirium, strangeness of manner, stupor, convulsions, and coma. Death may be gradual or sudden.

Treatment.—In cases in which the diagnosis can be reasonably made, the use of perchloride of mercury, iodide and bromide of potassium, and local counter-irritation or anodyne applications for the relief of pain, are indicated.

GENERAL PARALYSIS OF THE INSANE.

(*Paralytic Dementia.*)

Although mental diseases do not come within the scope of this work, it is desirable to describe this complaint, because it depends upon actual structural changes in the central nervous system, and because the paralytic symptoms are often the prominent features of the case for long periods of time, and may give rise to a difficulty in diagnosis from other purely physical conditions.

Shortly stated, the disease consists in progressive symptoms, partly of a paralytic, partly of a mental character, terminating in dementia and complete powerlessness, and dependent upon widely-spread anatomical changes in the brain, spinal cord, and nerves.

Ætiology.—It occurs very much more frequently in men than in women, and mostly in the prime of life—that is, between the ages of thirty and fifty. Syphilis is an antecedent in about three-quarters of the cases, and other factors are sexual excess, alcoholic indulgence, mental worry and overstrain, business anxieties, and sometimes injuries. Heredity seems to have much less influence here than in many other mental ailments.

Symptoms.—Considerable differences are seen in the grouping of the symptoms; in some cases the paralytic features are more prominent, in others the mental. The paralytic symptoms are sometimes more of a cerebral nature, at others more spinal, and they may for a time exactly resemble those of locomotor ataxy or disseminated sclerosis; the mental failure may be at one time marked by exaltation, at another by depression, but ultimately, in all cases, dementia supervenes. To begin with that which has long been considered the most typical case of general paralysis of the insane, the first thing noticed is often an alteration of a moral or intellectual kind; and this may be for some months or a year or two before anything more decided is observed. The man becomes careless or neglectful, tends to intemperance in drink, or spends money more freely than has been his wont without any justification; or he is irritable or restless, changed in his affection to his wife or family, or jealous without cause. The first indications on the physical side are generally tremor of the tongue or lips, or hands; the gait then becomes uncertain or tottering; but without any well characterised ataxy. More marked weakness of the tongue and lips causes defects of articulation. Many-syllabled words, such as "artillery" or "biblical," are confused, or some syllables are doubled or misplaced; other words are clipped short. The handwriting is uncertain and shaky, and letters are apt to be dropped out, or words left unfinished; and in turn all other finer movements, which have been attained by careful education, such as piano-playing and violin-playing, are lost. The reflexes are exaggerated. The pupils are often unequal, sometimes closely contracted; but the Argyll-Robertson symptom is not generally present. By the time that these forms of weakness have become pronounced, if not before, the mental deficiency has reached the stage of delusions, and in many cases these delusions are of an exalted kind, and ideas of grandeur fill the mind of the sufferer. These concern himself alone; they express what he is, what he possesses, or what he can do. He is the Almighty, or the king of England, or the prime minister; the most handsome or the most powerful man in the world. He has boundless wealth, hundreds of carriages, millions of gold watches, or countless wives. Sometimes the first indication of these ideas is revealed by his going to a shop and ordering useless quantities of expensive goods, far beyond his means. Restlessness of body and mind is also a characteristic at this stage.

There is then either a gradual transition to a *second stage*, or a more rapid change by one or more convulsions, from which the patient recovers considerably worse than he was before. His mental power is more deficient, his memory fails him, the lofty ideas may be present, but he is less influenced by them, and is more manageable. The muscular weakness is more apparent; it shows itself in the want of expression in the face, in the very imperfect articulation, and in loss of power in the arms and legs. Common sensibility is also diminished or lost; but appetite is often retained, and the patient is not infrequently fat.

He gradually becomes more feeble-minded, and, finally, the *third stage* of complete dementia, with loss of control of the bladder and rectum, is reached. He sits about, or is too weak to do more than lie in bed; he is subject to convulsions from time to time; the limbs may become contracted; and bed-sores will form unless the greatest care be taken. He dies from pneumonia or bronchitis; or swallowing is difficult, and he may be choked; or cystitis or bed-sores may lead to secondary blood-poisoning.

Varieties.—Some cases never show the stage of exalted ideas, but are *melancholic* from the first, and gradually pass into the final condition of dementia; others are *demented* throughout, without exaltation or melancholia. A *double* form is also spoken of, in which the stage of exalted ideas is followed by one of depression, and this again by one of exaltation. The early paralytic symptoms are sometimes distinctly spinal. The case presents, perhaps, for some years all the characteristic features of *locomotor ataxy*, with shooting pains, absent knee-jerks, ataxy, and Argyll-Robertson pupils; or of *lateral sclerosis*, with weakness and rigidity. Savage says this is not infrequent in women, single as well as married, and at a rather younger age than is common with other varieties. Or the features are those of *disseminated sclerosis*, with nystagmus, staccato speech, and oscillations of the limbs and trunk on movement; or *mixed* forms, suggestive partly of one, partly of another of these three diseases, may be present.

Some special conditions of the nervous system and nutrition may be mentioned. Fits or convulsions may occur at almost any period of the case; they may be slight or severe, passing off very quickly or leaving the patient comatose for some time; the convulsions also may be scarcely noticeable, or unilateral, or general. Amongst the optic symptoms the pupils have been already mentioned; but the optic discs are often unaffected, and ptosis and strabismus are rare. The muscles seem to present no constant changes either in nutrition or electrical reactions. The bones are brittle; the skin is often pale, waxy, and especially greasy; sometimes bullæ form, and a marked capillary congestion over the malar bones is common. There is a tendency to whitlows on the fingers, and to subcutaneous hæmorrhages, which sometimes lead to septicæmia.

The temperature is high in acute cases, and after convulsions, when it is often accompanied by free sweating. It is also raised by much bodily exertion or mental excitement, and by complications such as bed-sores and lung disease.

Duration.—This is variable; there are acute and chronic cases.

If the early symptoms of gradual mental change, and the early spinal symptoms, in cases beginning with locomotor ataxy or sclerosis, be excluded, the duration is not often more than two years.

Morbid Anatomy.—The changes are very variable, but the following are found in different cases:—Thickening of the calvarium, which is much marked by Pacchionian bodies; thickening of the dura mater, with false membranes (pachymeningitis); abundant subarachnoid fluid, with thickened or adherent membranes, the adhesion, when present, being more over the frontal, parietal, and temporo-sphenoidal lobes, and more on the upper than the lower surface; wasting of the convolutions, especially the ascending parietal, paracentral, and first frontal at its base; a violet-red colour of the cortex of the brain; in some cases much fluid in the lateral ventricles, with softening of the brain-tissue; in a larger number, a general hardening of the brain. In the spinal canal, the same changes may be found; pachymeningitis, or adhesion of membranes, or effusion of blood within the dura mater. The spinal cord is wasted, or presents the lesions of posterior, or of lateral, sclerosis. Microscopic examination of the brain shows increase of connective tissue, accumulation of leucocytes in the perivascular sheaths of the minute vessels, and alterations in the nerve-cells of the cortex, especially the pyramidal cells of the third layer. The nerves are also degenerated, but the sympathetic ganglia are, according to Savage, not appreciably affected.

Diagnosis.—*Alcoholism* may be mistaken for general paralysis, the tremor of the lips, tongue, and hands largely contributing to this; commencing *peripheral neuritis* might further complicate the case. But the close association of the symptoms with continued drinking, the absence of inequality of the pupils, and the improvement on prolonged abstinence would point to alcoholism. Mental failure, with definite cerebral lesions such as *tumours*, or the dementia following *apoplexy*, may give rise to difficulties. From the general physician's point of view it is important to recognise that various anomalous paralytic symptoms may be warnings of general paralysis. If a case is typically locomotor ataxy there is no special reason to anticipate mental trouble; but if the symptoms develop very rapidly or present unusual groupings, or if there are mixed symptoms not conforming to the ordinary types of the spinal-cord diseases, the mental condition should be closely scrutinised, and the possibility of general paralysis of the insane should be kept in view.

Prognosis.—In an undoubted case it is bad, death being the certain termination; but temporary improvement (remission) sometimes takes place, especially in the cases with exaltation of ideas.

Treatment.—This is, of course, in the highest degree unsatisfactory, as nothing seems able to stop the progress of the very widespread changes in the nerve-tissues. If the disease is recognised early, the patient should be at once removed from all sources of worry, anxiety of business, &c.; he should get change of scene, but should be kept under observation. Excess of every kind is to be avoided; and hitherto drugs have been found to be of little or no service except in ameliorating symptoms. Some, who believe that syphilis is the chief factor in its causation, advocate iodides and mercury; but the same line of treatment has not succeeded in the analogous case of locomotor ataxy.

FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.

EPILEPSY.

Epilepsy is a disease in which there are attacks of sudden loss of consciousness with or without convulsions, independent, as far as our present knowledge goes, of any demonstrable lesion of the brain, or peripheral irritation, or blood-poisoning. Although the name is commonly associated with the idea of convulsions, and these indeed occur in the most typical and severe forms of attack, still it is important to note that coma is almost invariably present with the convulsions, and in many slighter attacks there is no convulsion at all. The second part of the definition excludes those convulsions which may arise from organic cerebral lesions, such as tumour, or from Bright's disease, or from anæmia. These are often called *epileptiform* to indicate their close relation to the *epileptic* convulsions now under consideration.

Epilepsy, then, is a functional disorder of the brain, a functional neurosis, or spasmodic neurosis, as it has been named by some; and its recognition, to a certain extent, depends on the absence of any other symptom from which the existence of structural lesions or diseases likely to cause convulsive phenomena could be inferred.

Ætiology.—It is slightly more frequent in females than in males, and very much more often begins in early life (Gowers says 75 per cent.) than in middle or advanced age, though it may be common enough in the latter class, simply because it is in a great majority of cases incurable, yet not fatal. Thus, beginning at an early age, it may continue throughout the life of the individual, who eventually dies from other causes. Among the predisposing conditions, inheritance has the greatest importance; and this shows itself partly by the appearance of epilepsy in the children of epileptics, but very largely by its occurrence in the offspring or other blood-relations of those who have suffered, not from epilepsy, but from some other serious disorder of the nervous system, such as insanity, hypochondriasis, hysteria, and marked neurotic weakness, or nervousness. Dipsomania in the parents may occasionally be a contributing cause. When epilepsy is not inherited, but acquired, alcoholic indulgence, sexual excesses, and possibly masturbation, may be the predisposing causes. But the first two are not so likely to be in operation at the period of life when epilepsy generally begins, and the last more often leads to hysterical conditions simulating epilepsy—hysteroid epilepsy. The more immediate causes of a first epileptic attack, which may be the beginning of a lifelong series, are fright, mental anxiety or excitement, injuries to the head, fevers such as scarlet or enteric, and the presence of worms in the intestines.

Epilepsy occurs in two well-marked forms, described as major and minor attacks—epilepsia major and epilepsia minor, or, in the terms of French writers, *haut mal* and *petit mal*. The major attack consists of the fully developed fit, with coma and violent convulsion, which will be immediately described. The minor attack consists of a momentary loss of consciousness, with either no convulsion at all, or at most a little twitching or some other slight sensory or motor disturbance; or rarely, motor disturbance without unconsciousness.

Major Epilepsy.—This occurs in several stages—(1) aura; (2) unconsciousness and tonic contraction; (3) clonic convulsion; (4) recovery. The *aura* (or breath, from the sensation of air passing up the limb to the head, which constitutes one form of this symptom) is any sensation or motion experienced by the patient while he is yet conscious, mostly of very short duration, and terminating abruptly in loss of consciousness and convulsion.

1. There is a great variety of auræ, which may be felt in almost every part of the body—in the limbs, face, and head, in the viscera, and the organs of the special senses. They have been classified as sensory, motor, vasomotor, and psychical. The following may be mentioned: tingling and numbness in the arm, leg, face, or tongue; twitching or spasm in the same parts; loss of vision, or visual hallucinations, such as flashes of light, or colours, or definite objects; hallucinations of sound, noises, &c.; unpleasant odours or tastes; sensation of choking, nausea, vertigo, epigastric pain; flushes of heat, coldness, palpitation of the heart; an indefinite sense of fear or anxiety; running and jumping, or other co-ordinated movement. Auræ of sensation and motion are

mostly unilateral, but may be bilateral; the arm is more often affected than the leg, and facial auræ mostly consist of spasm. Visual auræ are much more common than auræ of the other special senses. Sometimes a vague sense of fear may last some time before the occurrence of the actual fit; but, as a rule, the aura is of momentary duration. In about half the cases it is entirely absent.

2. The fit itself commences with sudden unconsciousness; if standing or walking, the patient often falls suddenly forwards, or seems to be thrown violently to the ground, sometimes with an involuntary cry, shriek, or groan—the epileptic cry. He is then found to be in a state of tonic convulsion, the back rigid and slightly arched, the legs extended, and the head drawn backwards or rotated to one side. The face is often pale at first; the pulse is quick, but sometimes it cannot be felt, and this was attributed by Fagge to compression of the artery by muscular contraction. The general tonic contraction fixes the chest, and respiration is stopped, so that gradually the face becomes more and more dusky, and eventually quite cyanosed. The tonic stage lasts from three to thirty or forty seconds, and then passes into the stage of clonic convulsions.

3. Twitchings begin in the face, the eyelids, and the side of the neck, and quickly extend to all the muscles of the body and limbs. There is a rapid succession of to-and-fro movements, of alternate flexion and extension in the limbs, of opening and shutting of the eyelids and of the jaws, lateral deviation of the eyeballs, and perhaps of the head; the tongue is pushed forward, and may be caught between the teeth; saliva is freely secreted, frothed in the mouth, and escapes from the lips mixed with blood from the bitten tongue. The face becomes livid, or almost black, and the lips and features are swollen. Urine, fæces, and, in men, semen may escape during this stage, and the violent contraction of the muscles may even cause dislocation of the shoulder. The patient is, of course, quite insensible; the conjunctivæ do not respond to a touch, the pupils are dilated or oscillate.

4. The clonic stage lasts a few minutes, rarely more than five or six, and then the convulsions gradually subside—they become less frequent, and interrupted by pauses of some seconds; the breathing becomes easier, the frothing at the mouth ceases, and the face gradually assumes a more normal colour. Finally, the patient remains simply comatose, and the coma passes into natural sleep, or consciousness is recovered rather suddenly soon after the cessation of the convulsions.

The reflexes are mostly absent for a short time after the attack, and then for a time the deep reflexes may be increased (Gowers). The urine may contain a trace of albumen or sugar; petechiæ may be seen under the skin from rupture of blood-vessels during the stage of venous congestion ; sometimes there is a transient hemiplegia; or vomiting; or serious mental disturbances, such as delirium, which is often of a maniacal kind.

The mechanical injuries from which the epileptic suffers will, of course, remain after the fit, and may give valuable indications in cases where the fit has not been seen—for instance, in nocturnal epilepsy. These are the bitten tongue, petechiæ on the skin, a dislocated shoulder, and, in other cases, various cuts, wounds, or bruises from the falling of the patient upon the ground or against unyielding objects.

Minor Epilepsy.—This consists, in a large number of instances, of little more than a sudden unconsciousness: in the midst of talking, perhaps, the eyes become fixed, the pupils dilated, the speech incoherent, and the patient is obviously unconscious of what is going on around him; he may, if at meals, put his fingers in his plate or his cup, or commit some other irregularity that he would not do if conscious. The condition lasts a few seconds, and then he becomes conscious, and goes on with what he was doing, or perhaps recognises that there has been a blank, or feels giddy, or has headache, and is glad to lie down for some time Sometimes giddiness is the most marked feature of the attack, and in other cases a sensation in one or other part of the body, or a spasmodic movement, which may be quickly followed by temporary unconsciousness, though the former will seem to the patient the chief feature of the attack. These have a close resemblance to the auræ of the major attacks, and Gowers enumerates as occurring in minor epilepsy sensations in the epigastrium, hands, head, nose, eyeballs, and cardiac region, olfactory and visual sensations, jerks in the limbs, head, or trunk, sudden tremor, sudden screaming, sudden dyspnœa, mental conditions, such as a sudden state of fear, &c.

Post-epileptic Conditions.—The *petit mal*, even more than the major attack, is liable to be followed by certain post-epileptic conditions. One of these consists of various automatic actions, of which the patient is then and afterwards entirely unconscious. He may thus commit acts of violence, rushing about and striking all that he comes near, or a woman may kill her child, or one may appropriate things that do not belong to him. Trousseau records the case of the judge who relieved his bladder in the corner of the room, without any consciousness of the act. These cases have great medico-legal importance, since the occurrence of epileptic fits may be entirely unknown, and the criminal acts may be attributed to wilful and conscious violence. Sometimes these attacks are distinctly maniacal in their character (epileptic mania), and the automatic actions are accompanied with much mental disturbance, such as terror, violent passion, and hallucinations. In girls, boys, and young women, the minor attack may pass into a hysteroid condition (*see* Hysteria). These phenomena have been regarded by some writers as replacing the fit in many cases, but Gowers is inclined to regard them as post-epileptic.

Though we can generally distinguish between the major and minor attacks, there are attacks which present intermediate characters. The two forms are often only different phases of the disease in the same person; thus it is not uncommon for children to suffer first from minor epilepsy, and as they get older to develop the major attacks. They may both occur in the same patient alternately, or more or less irregularly.

Course of the Disease.—The frequency of epileptic attacks varies considerably in different cases, and at different periods in the same case. Thus, there is generally an interval of one or more months between the first and second attacks, but with the progress of the disease the intervals often become shorter, and the fits may be as frequent as two or three in a week, or even several in a day. In some cases two or three fits occur in quick succession, or at short intervals, and the patient is then spared for a long time. A severe fit is much more likely to be followed by a long interval than a slight one. Probably alcoholic indulgence, injudicious feeding, mental or physical over-exertion may increase the frequency of the fits. In some female epileptics the attacks come on with each menstrual period. As will be shown, the frequency of the attacks can be very greatly diminished by the use of certain drugs. In rare instances the patient has a series of fits, extending over some hours, or one or two days, and never recovers consciousness in the intervals between them. This is called the *status epilepticus.* The heart beats violently and rapidly, the respirations are quick, twitchings occur in the intervals of the convulsions, the temperature often rises to 105° or 107°, and the patient may die collapsed, or may become delirious. The condition is often fatal.

Health between the Attacks.—This depends a good deal upon the frequency of the fits. Where these are not numerous, the individual may enjoy excellent health. Many epileptics are strong, hearty, and vigorous, never ailing at all except when the subject of the periodical attacks. When, however, the fits are very frequent, or the disease has lasted a long time, the mind generally suffers, the patient becomes dull and irritable, the memory is deficient, and intellectual processes are slower; and in some cases actual imbecility is the result. In children, sometimes, even after a few fits, permanent imbecility or mania may be developed.

Death from epilepsy is by no means common, and, except in the case of the rare status epilepticus above described, it is mostly the result of some injury to which the patient is exposed during the fit. Thus, during a fit he may be thrown from a height, or fall into water and be drowned, or be choked by food, or he ma be smothered in bed by his face being buried in the pillow, or h may die later from injuries received by a fall into the fire or fro a carriage or bicycle.

Pathology.—The only anatomical conditions that have at a frequently been found in fatal cases are thickening of the bon of the skull or of the cerebral meninges in old cases, the conge tion of the viscera in those who have died in the actual fit. I some cases nothing abnormal can be recognised.

Many considerations render it probable that the seat of th disturbance which brings about the epileptic fits is in the corte of the brain. These are—first, the association of coma with th convulsions; secondly, the existence of cases of masked epileps in which the symptoms are mainly psychical, such as epilept mania and delirium; thirdly, the fact that definite lesions of th cortex, such as tumour or gumma, produce convulsions identic in character with those of epilepsy; fourthly, the generally un form spread of the convulsions from face to arm, and arm to le corresponding to the relative positions of the motor areas for tho parts on the surface of the brain—a result which may be observe not only in the idiopathic epilepsy of man, but in the epilept convulsions produced by experimental irritation of the brain i animals; fifthly, the fact that some cases of epilepsy begin wit definite lesions of the cerebral cortex—*e.g.*, as the result of blows sixthly, the cases occasionally recorded in which epileptic fits hav ceased after the development of disease in the internal capsule.

The nature of the initial change is altogether doubtful. Th view of some authors, that it is due to a sudden vasomotor spas constricting the vessels, is supported by the fact that, experimen ally, convulsions can be caused by producing anæmia, and by th occurrence of convulsions in anæmia from loss of blood in man but these do not suffice to prove it. One cannot say much mor at present, than that it is an unstable condition of the nerv tissue, permitting occasional discharges.

Diagnosis.—Epilepsy is with no great difficulty recognise when actually seen, but one is often called upon to prescribe fo fits which only occur at times when the physician cannot witnes them; and it is not always easy to come to a right conclusio from the descriptions of friends. The major attacks have to b distinguished from attacks of hysteria, and from simulated fits minor epilepsy, from attacks of syncope. In hysterical attack the movements are more purposive, and more automatic—that i to say, they are not mere alternating contractions and relaxation of antagonistic muscles, but more combined movements, apparently made with a purpose or object. Thus, the patient ma dash her head repeatedly against the floor or the bed; and, i efforts are made to restrain her, she will struggle to throw o those who are holding her, or will bite and clutch at those near her. The facial muscles may twitch, and some saliva may come from the mouth, but it is not tinged with blood, and the tongue is not bitten. The face is generally red or pale, sometimes rather blue about the lips, but it never presents the intense cyanosis of epilepsy. The eyelids often quiver, and resist attempts to open them. The fit of hysteria is of long duration, lasting half an hour or longer, whereas that of epilepsy is over in a few minutes. The mere fact of unconsciousness is not conclusive, as the events of a hysterical fit are not in the least recalled by the patient. But, in hysteria, there is an automatic response to sensory and auditory impulses, while in epilepsy the patient is, for the time, absolutely senseless.

In *Jacksonian epilepsy* (*see* p. 310) the unilateral localised convulsion is primary, and loss of consciousness is either absent or secondary.

The *malingerer*, who attempts to excite sympathy as a sufferer from epilepsy, can, with a little care, generally be detected. He is careful to fall so as not to hurt himself, whereas the epileptic is thrown down suddenly, and if in the street will probably strike his head or face, or will fall in the road, not making any effort to save himself. The malingerer is red in the face, rather than pale or livid; his skin perspires from the exertion, his pupils are not dilated, and are sensible to light. The fact that he has not lost consciousness may be tested in various ways: by touching the conjunctiva, when the eyelids will close, though he will probably resist attempts to raise the upper eyelid; by applying snuff to the nostrils; by producing some very painful impression, as by forcing one's thumbnail under that of the malingerer.

Minor epilepsy, or *petit mal*, is distinguished from *cardiac syncope*, or simple fainting, by its occurrence under circumstances not conducive to fainting, by its suddenness, and by its rapid recovery, followed by mental confusion, rather than physical prostration. The occurrence of spasm or of any warning sensation other than the feeling of faintness is in favour of epilepsy. Syncope comes on more slowly, and is recognised as a gradually increasing faintness by the patient. Still, this may happen as a warning sensation of *petit mal*. To distinguish other forms of *giddiness* from the vertiginous form of minor epilepsy, one must remember that ordinary vertigo is not accompanied by loss of consciousness, and that in the aural form, or Menière's disease, there are persistent deafness and tinnitus.

When it has become certain that, in any case, the convulsions are really epileptiform in character, it has yet to be determined that they are not due to tumour of the brain, peripheral irritation, or the uræmia of Bright's disease, before we can pronounce the disease to be epilepsy. In a great number of cases of idiopathic epilepsy, the long history of recurring convulsions with no associated symptoms will serve to distinguish it, whereas in *local disease of the brain* there will probably be other indications, such as headache, vomiting, optic neuritis, or local paralyses. In *Bright's disease* the convulsions are epileptiform, but the patients, as a rule, show good evidence of their state of health, in albuminuria, high tension of pulse, hypertrophy of heart, preceding uræmia, or œdema; the fits are often ushered in by drowsiness and muscular twitchings, are of much longer duration, and recur frequently in the same day with intervals of drowsiness or semi-coma. Any source of *peripheral irritation* should be inquired into, such as a decayed tooth, intestinal worms, and in children dentition, phimosis, constipation of the bowels, pins in the clothes, &c.

It remains to be mentioned that nocturnal attacks of epilepsy may be for a long time unrecognised, if they are not actually witnessed by any one. They may be suspected if a boy or girl, not suffering from nocturnal enuresis, and beyond the age at which that is usual, unexpectedly wets the bed; or if there are petechiæ on the face or body, or a sore tongue which the patient cannot account for, or headache.

Prognosis.—Epilepsy rarely recovers without treatment, and the hope, so often entertained by the patient's friends, that attacks beginning in youth will cease with the development of puberty, or with the appearance of the menses, is nearly certain to be disappointed. They can, however, be very markedly controlled by treatment, and are generally the more amenable the later in life they have begun. Gowers states that the prognosis is better if the fits occur only during waking, or only during sleeping hours, and not under both circumstances; if there is no considerable mental change; if the attacks are only of the major kind, and not both major and minor; and that it is better if there is an aura than if there is none.

The effect of the fits upon the mental condition of the patient is, as a rule, directly in proportion to the duration of the illness and the frequency of the attacks.

In themselves the attacks are not dangerous, except from accidents to which they may lead, such as falling into water, or into the fire, or from heights, and especially from being smothered in bed during a nocturnal attack. The status epilepticus is often fatal, but is a rare condition.

Treatment.—The object of treatment is to reduce the frequency, or prevent the recurrence of the fits. The management of a patient during a fit has also to be considered.

In the Interval.—Something may be done in many cases by careful attention to diet, and other hygienic matters. Food should be light and digestible, large meals should be avoided, and especially heavy suppers just before going to bed. Stimulants should only be taken in moderation. Regular but not exhausting exercise, and an occupation that does not involve danger to life, if a fit occurs, should, if possible, be obtained. Children with epilepsy should be educated suitably to their position in life, but need not be forced into competition.

Of medicines, the most useful are the bromides of potassium, sodium, and ammonium, which have a very powerful influence upon the course of the fits in the majority of cases. The potassium salt is the one most generally employed, and should be given in doses of from 10 to 30 grains three times a day. For milder cases and young subjects the smaller dose may suffice. Severe cases will require the larger dose, or even more. A combination of the salts is preferred by some, such as the bromides of potassium and sodium, or of sodium and ammonium, or of all three. Lithium bromide has also been used. The dose of the combined salts may be the same as that of either given separately. Whichever salt is used, it must be continued for several months, or years. Its effect is generally to diminish the frequency or severity of the fits, so that they occur at intervals of months, instead of every week; if the bromide is left off, the fits again become more frequent, and consequently the patient is glad to continue the use of the drug as a part of the daily routine. Even if the fits cease entirely for several months or a year, the bromide should be continued for two years after the last fit, and then in gradually diminishing doses for another year.

Large doses of the drug are best taken in plenty of water, and the eruption of acne which sometimes occurs from its use may be prevented by adding three or five minims of liquor arsenicalis. Very large doses of bromide are also inconvenient in another way, making the patients dull and lethargic, with muscular weakness and cold extremities. This condition is known as *bromism*, and is liable to be induced by doses larger than half a drachm three times a day. If it occurs the drug must be diminished in quantity, or stopped altogether for a time.

Several other drugs have been used for epilepsy; none is so good as the bromides, but they may be used in combination with them, or may replace them from time to time, for instance, when bromism has been induced. They are belladonna, zinc sulphate, oxide, or lactate (up to 10 or 15 grains three times a day), iron, borax (15 to 30 grains), digitalis, and cannabis indica. Various combinations may be tried, and Gowers speaks well of a mixture of belladonna, lactate of zinc, and bromide of potassium.

During the Attack.—In cases where there is a definite aura the attack can sometimes be arrested. If the aura consists of a sensation in the hand, which gradually proceeds up the arm, the fit may possibly be checked by vigorously rubbing the part, or by tightly constricting the arm above the seat of the sensation, thus preventing, as it were, its progress to the centres. Where this is successful the patient may wear a cord looped round the upper arm, with one end conducted down his sleeve to the wrist, so that by pulling upon this end he can at once constrict the arm. Other patients ward off fits by lying down on feeling the aura, or by answering the indication afforded by the aura, as in the case of a patient of Strümpell's, whose fits were preceded by a sensation of tenesmus, and might be sometimes checked by her going to stool. Occasionally the inhalation of nitrite of amyl will prevent the further development of an attack. When the fit has really begun little can be done in the way of treatment; but the patient can be protected from some of the results of the convulsions. As a rule he must lie where he falls, unless this is in itself a position of danger (a pool of water, or the fire), but he can be prevented injuring himself against surrounding objects; his collar, necktie, cuffs, and other tight bands, should be at once loosened; and a piece of cork, gutta-percha, or fire-wood, should be held between the teeth to prevent the tongue being bitten. Fagge gives a word of caution with regard to false teeth, which may become loosened in a fit, and be impacted in the pharynx. They should be, therefore, always removed at night.

For the *status epilepticus*, the inhalation of nitrite of amyl is strongly recommended by Sir Crichton Browne; other measures are chloroform and ether inhalations, chloral in 15-grain doses every four hours, small morphia injections ($\frac{1}{10}$ grain), a moderate venesection, and ice to the spine.

INFANTILE CONVULSIONS.

Convulsions occur with much greater readiness in infancy than in later periods of life, and under somewhat different circumstances. The higher centres are less developed and exert less controlling power over the lower. The circumstances under which convulsions generally occur are the following:—(1) The onset of acute diseases, such as scarlatina, measles, and pneumonia; the convulsions here seem to take the place of the rigor of adults. (2) Local diseases of the brain, of which acute meningitis is the most frequent; but tubercular tumours, chronic hydrocephalus, and lesions following otitis may occasionally be the cause. (3) Great exhaustion, as after prolonged diarrhœa, or diarrhœa and vomiting; the resemblance of this condition formerly called hydrocephaloid disease, to acute meningitis, has been already mentioned (p. 300). (4) Venous congestion of the brain, such as may be caused by an attack of whooping-cough, which sometimes terminates in general convulsions. I have seen a child cry itself into convulsions at once if its mother left it, holding its breath, and becoming more and more livid, until the fit broke out. Convulsions which not infrequently occur at the end of a pneumonia may sometimes belong to this group. (5) Rickets is now held to be responsible for the great majority of cases of infantile convulsions not included in the above groups. Often the fit is induced by some peripheral irritation, such as indigestible food; intestinal worms, especially lumbrici; cutaneous irritation, such as pins in the clothing, instanced by Trousseau; or the process of dentition. Perhaps too much has at different times been made of teething as a cause of convulsions, since delayed dentition is a constant result of rickets, and so would co-exist with convulsions in a large proportion of the cases. Sometimes no exciting cause for the fit can be discovered. (6) Some infantile convulsions must be regarded as really epileptic, since epilepsy may begin in infancy; especially those must be so regarded which commence in early childhood—*e.g.*, at two or three years, when the influence of rickets is beginning to wane.

Of these six groups, it is especially the last two that are usually considered as infantile convulsions proper, or *eclampsia infantum*, the convulsions in the other cases being more definitely symptomatic. Convulsions in children may closely resemble the epileptic fit of adults; but in a large number of instances, they are less complete. They often begin with a short tonic stage; the eyes are turned to one or other side, the pupils are dilated, the head is drawn back, and the arms and legs are rigidly extended. The face may be at first pale; but the lips soon become livid. Twitching then begins in the lips or eyelids, and extends to the whole body, which may be thrown into violent clonic convulsion. The fit lasts a few minutes, and is followed by recovery; or there is a succession of fits, alternating with coma, during which slight twitchings of the facial muscles or extremities may take place. Often the convulsion is very much slighter, and consists of little more than deviation of the eyes, or squinting, or fixation of the chest with commencing lividity of the lips, or the convulsive closure of the glottis, known as *laryngismus stridulus* (*see* Diseases of the Larynx); or the hands are extended and rigid, with the thumbs turned into the palms, or the hands and feet are disposed in the manner characteristic of tetany (*see* p. 349). Convulsion may be followed by temporary hemiplegia in children as in adults. Strabismus is another occasional result. Finally, convulsions in children are not infrequently fatal.

Twitchin

Diagnosis.—The recognition of infantile convulsions is not itself difficult. It is necessary, however, to determine upon what they depend. If it is a first fit the possibility that it is the onset of an *exanthem* must be remembered; the temperature should be taken, and the child carefully watched for a time afterwards. Fits due to *cerebral disease* are more likely to be unilateral, and may be accompanied with other symptoms, such as headache, vomiting, retracted abdomen, or optic neuritis. In other cases the indications of rickets must be sought for in the beaded ribs, the enlarged epiphyses, open fontanelle, and delayed dentition; and careful inquiry should be made after some source of irritation, such as unsuitable food, and others above-mentioned.

Treatment.—This again consists of the treatment of the fits, and the means to be taken to prevent recurrence.

When a fit occurs it is usual to place the child at once in a warm bath. If the bowels have not been recently opened, or if there be reason to suppose the ingesta are causing irritation, a grain or two of calomel may be placed on the tongue. If the fits are very violent and continuous, chloroform may be cautiously administered. It will promptly check the convulsions; but they will probably return soon after it is withdrawn, when it may be again given for a few minutes. When the child recovers sufficiently, 5 grains of bromide of potassium may be given; or if the fits are continuous, it may be given in somewhat larger doses (7 or 10 grains) by the rectum. Chloral may be combined with it to the extent of 3 to 5 grains.

To prevent the recurrence of the fits, one must deal with the predisposing condition, and with the special susceptibility to convulsion. If an exanthem, meningitis, cerebral disease, or whooping-cough is the cause of the fit, the disease must be dealt with as advised elsewhere, and the fits accompanying such illness are very little amenable to special treatment. The frequency of convulsions in rhachitic children can be much influenced by treatment suitable to rhachitis, such as regulation of the food, administration of cod-liver oil, and general hygienic improvement, together with the use of potassium bromide in doses of 2 or 3 grains three times daily. Similarly, the cases that are more allied to epilepsy should be treated by the regular use of the bromides.

MIGRAINE.

(*Megrim, Sick Headache, Hemicrania.*)

This complaint consists of recurring attacks of headache, preceded by certain abnormal sensations, and often associated with nausea and sickness.

Ætiology.—It is undoubtedly hereditary, and, like epilepsy, it may have hereditary connections with other neuroses; or it may occur in those who inherit gout. Though it may begin in early childhood, it commonly first appears about the age of puberty, and lasts through the greater part of life; it rarely commences at an advanced age. It is, perhaps, more frequent in women than in men. The attacks are more likely to occur in those whose occupations are sedentary, or whose work is chiefly mental; and it is disposed to by various improper hygienic conditions. The immediate cause of an attack is often some disturbance of digestion by a large meal, or indigestible food, or by constipation, or it is some exceptional mental or bodily fatigue, or worry or anxiety. Overstrain of the eyes, as in long reading, or in sight-seeing (theatres, picture-galleries), is a not uncommon cause, and especially if there is any uncorrected error of refraction; thus, hypermetropia, astigmatism, and muscular asthenopia, which are frequently the cause of frontal headaches, occasionally lead to definite attacks of the special kind of headache known as migraine.

Symptoms.—A complete attack consists of the sensory phenomena and the succeeding headache; but sometimes the attack consists only of headache, and at others the sensations are experienced alone without being followed by pain in the head. The most characteristic commencement of migraine is by a visual sensation consisting of half-blindness or *hemianopia*. In a great number of cases it takes place as follows: the patient may be apparently in perfect health when he notices that he is unable to see what is directly in the centre of the field of vision, but has to shift his head a little for the purpose; or he sees at once that the centre is occupied by a bright spot. In a few minutes the area of blindness enlarges, and if he turns towards a dark part of the room, or to a clear surface, like the ceiling, he will see a small circle of sparkling colours, having a zigzag or vandyked pattern. From minute to minute the circle grows larger and larger to one or other side and away from the centre, opening out so as to form a horseshoe, which obscures the field of vision as it spreads outwards, while vision returns in the centre, and progressively improves. The outer margin of the horseshoe consists of a zigzag line of colours; within this is the blind area, full of movement as of a boiling fluid; within this again, the recovered area of vision. Generally, in half an hour from the beginning the horseshoe has reached the periphery of the field; every object is now visible, though, perhaps, with a sensation of oscillation or quivering. By closing each eye alternately it will be seen that the sensation is not due to an affection of either of them, but to a disturbance of the brain which must be situate beyond the optic chiasma on the side opposite to the blind part of the field. This curious spectrum has been described as *teichopsia*, or fortification figures. The visual phenomenon is not always so definite as this—it may consist only of flashes of light or half-blindness without spectra.

Much more rarely other sensory disturbances occur, and they generally follow the hemianopia in the course of half an hour or an hour, or they may occur without it. They consist of tingling sensations in the limbs, face, tongue, or other parts; they may begin in one finger, spread then to another, and so to the hand or up the arm, and to the face or throat, the part first attacked recovering as the others are invaded. These sensations are mostly unilateral; they may be followed by transient weakness. Aphasia is another disturbance which may occur in the course of migraine; words are forgotten or misplaced, the condition resembling that of one who has just had too much wine. Aphasia is commonly associated with a spectrum on the right side of the field, and if tingling coexists that also is on the right side. Thus, the cerebral disturbance is on the left side, as it is in cases of aphasia from embolism or hæmorrhage—at least, this is true of right-handed persons. In some cases the only disturbance preceding the headache is of a mental kind; there is depression, languor, or fear of impending evil; and these feelings may last from half an hour to two or three hours.

The more distressing feature of the illness is the headache which comes on sooner or later after the preceding sensations, generally as these are declining; though I know one case where it nearly always occurred the day after the spectrum, with a perfectly healthy interval between them. The headache is of all degrees of intensity; so slight as not to interfere with even mental work, or so severe as entirely to unfit the sufferer for any action whatever. It often lasts a whole day, and in the severer forms it increases gradually, until when at its height nausea and vomiting occur, and a certain amount of relief is afforded to the pain. After this it quickly disappears, or it subsides more gradually, or it continues till the patient seeks his bed at night, in which case he will probably awake the following morning cured, but with some sense of weakness or fatigue. The pain is often unilateral (*hemicrania*), but it may begin on one side and change over to the other, and even return again to the side first affected, or it may affect both sides at once; the side first affected is generally that which is opposite to the visual spectrum. The pain may begin in the frontal, temporal, parietal region, or behind the eye, often at a very limited spot; thence it may spread in different directions, or become general. It is often boring in character, and aggravated by movement, light, or sound; in bad cases the patients are only comfortable in the recumbent posture. With this the face is pale, and drawn, the hands and feet cold, the pulse feeble, small, and slow. Only in some cases it appears that as the headache continues these conditions are reversed, the face flushes, and sweating occurs. German writers, indeed, describe two varieties of migraine—*Hemicrania sympathetico-spastica* and *hemicrania sympathetico-paralytica*, in the first of which the affected side of the head is said to be pale and cold, with contracted arteries, dilated pupil, and increased secretion of saliva, while on the affected side in the latter there should be flushing, a hot red ear, contracted pupil, throbbing temporal arteries, and unilateral sweating. But these forms can by no means always be recognised.

The attacks of migraine recur at intervals of a few days or two or three months, an interval of three or four weeks being more common. Particular attacks may be determined by the special causes enumerated, but sometimes it is impossible to find out what has induced the disturbance. The duration of the disease is often life-long, although it sometimes becomes less frequent, or disappears altogether, after the age of fifty. Cases have been observed in which attacks of migraine have become less or ceased on the appearance of other neuroses, such as epilepsy, asthma, or spasmodic croup; and a similar relation to gout has been also shown to exist.

Pathology.—The popular idea that this is a gastric or "bilious" disorder is perpetuated by the use of the term sick headache, by the vomiting of bile which sometimes occurs, and by an attack being occasionally induced by an injudicious meal. But the latter cases form a very small proportion of the whole, and it is clear, from the preceding visual and other sensory phenomena, that it is quite early or primarily a cerebral lesion. Several authorities have advocated the vasomotor theory of its origin. Latham supposes that the earlier phenomena are due to a contracted condition of the vessels, and the headache to exhaustion and subsequent paralytic distension of the same. The division into two classes of cases, according as the vessels of the head and face are contracted or dilated, has been already referred to. But neither of these views can be well maintained, and it is much more probable that the sympathetic disturbances are secondary phenomena, and that the primary change is a disturbance of the cells of the cerebral cortex. Gowers points out that the disturbance is double; the blindness is due to inhibition, the spectrum to discharge. The inclusion of the disease under nerve-storms (Liveing) or paroxysmal neuroses (Fagge) does not give much explanation. In any case it is certain that no anatomical change is present as a cause of the disease.

Diagnosis.—The association of the headache with the various sensory disturbances, and its recurrence at intervals, are generally sufficient to distinguish the complaint. The headache of brain-disease is either continuous, or, if it remits, the intervals are shorter, and the attacks longer than those of migraine. Where the visual spectrum is present it is generally quite characteristic. Epilepsy with a visual aura may be confounded with it; but the aura of epilepsy is of very short duration, while the spectrum of migraine mostly lasts from twenty to thirty minutes. There is no loss of consciousness in migraine.

Prognosis. Under treatment much improvement may be observed both in the frequency, and in the severity of the attacks; though the disease commonly continues for years. It is, however, not dangerous to life, and there is no evidence that sufferers from migraine are more liable than others to hæmorrhage, thrombosis, and other diseases of the brain.

Treatment. *In the Interval.*—The patient should be placed under the most favourable hygienic conditions, including a carefully regulated diet, the avoidance of constipation, exercise without exhaustion, pure and bracing air, exemption from excessive brain work or mental worry. To these may be generally added the use of tonic remedies, such as iron, quinine, strychnia, arsenic, and cod-liver oil. Ocular defects, if any, should be remedied by suitable spectacles. Seguin, of New York, strongly advocated the use of cannabis indica to prevent the recurrence of attacks; it is best given twice a day in pills containing ⅓ or ½ grain of the extract, and these should be continued, like any other treatment employed with this object, for at least six months, and, if necessary, for longer. Bromide of potassium is uncertain in its action; it is most successful, according to Gowers, in cases in which the face flushes, or is unchanged in colour. For cases in which pallor occurs he recommends nitro-glycerine to be taken two or three times daily after food, in doses of $\frac{1}{200}$ to $\frac{1}{150}$ of a minim; he suggests that by frequently flushing the brain with blood the nutrition of the parts concerned may be improved.

In the Attack. If the headache is severe the patient should lie down in a darkened and quiet room, with a cold water compress on the head, and hot water bottles to the feet if there is a tendency to collapse. He may take soda water, or suck ice; there is at first little desire for food, but after a time some soup or beef-tea may be beneficial. Antipyrin is a valuable remedy in sick headache, and may be given in doses of 5 to 15 grains. Phenacetin [illegible] has a somewhat similar action. Many people find relief from strong coffee, or guarana, a substance which contains caffeine in greater proportion than either tea or coffee. It may be [illegible] doses of 15 to 30 grains mixed with water, [illegible] half an hour.

[illegible] doses bromide of potassium (20–30 grains), chloral [illegible] butyl-chloral hydrate (10–15 grains), or Indian hemp in doses of 10 minims of the tincture, or ½ grain of the extract, may be tried; of these, the first is most likely to be useful. Nitrite of amyl and nitro-glycerine on the one hand, and ergot on the other, have been given to influence the vaso-motor system, and have occasionally done some good, but they cannot be depended upon. Locally, besides cold applications, various anodynes have been employed with varying success, such as ether or bisulphide of carbon on cotton wool covered by a watch-glass, extract of belladonna, diluted ointment of veratria, and menthol.

VERTIGO.

This is a disorder of the function of equilibrium of the body, by which is produced a sense of unsteadiness or of movement of the body in one or other direction, or a sense of movement in surrounding objects, or an actual movement of the body itself. The term is more or less synonymous with *giddiness* or *dizziness.*

It is of very different degrees. It may amount only to a slight instability or unsteadiness. Sometimes the patient seems to fall forward, or turn round, when he actually does not move; in other cases, surrounding objects appear to be moving round, or up and down; or the sense of movement of the body and of surrounding objects may occur together. Often the patient reels and staggers, takes hold of a chair, table, or railing for support, or, failing that, may fall to the ground.

The equilibrium of the body seems to depend on various centripetal impressions, and upon the integrity of the middle lobe of the cerebellum. These centripetal impressions are derived from the skin, from the muscles, especially of the lower part of the body. and from the semicircular canals of the labyrinth of the ear. A knowledge of the position of the head and eyes also contributes to equilibrium, and this is probably derived from active innervation. If any one of these centripetal impressions is deficient, vertigo may result, and one of the most common causes of vertigo is a lesion of the labyrinth, producing what is known as *labyrinthine* or *aural vertigo.* Experiments on animals have shown that lesions of the semicircular canals will produce vertigo, the direction of the movement being determined by the canal injured, —whether horizontal, transverse, or vertical, whether right or left; and by the nature of the injury—whether irritative or destructive. But vertigo in man is caused not only by primary disease of the labyrinth, such as hæmorrhage or congestion, but by diseases of the middle ear, and of the meatus, such as inflammation of the tympanum, obstruction of the Eustachian tube, and collections of cerumen in the meatus; and even by syringing the ear, when the membrana tympani is perforated. Probably the effect is brought about by these morbid conditions altering the pressure of the endolymph in the labyrinth.

It is, however, the internal ear which is affected in the great majority of cases; the patients suffer, at the same time, from tinnitus, deafness, and vertigo; and the deafness is not due to imperfect conduction through the middle or external ear. It was in some very severe forms of aural vertigo that Menière called attention to the association of the symptoms, and the presence of disease in the semicircular canals; hence the term *Menière's disease* has been employed, and has been extended by most writers to every form, however slight, of labyrinthine vertigo. In well-marked cases there is constant tinnitus, with more or less deafness, of which, however, the patient may himself be unconscious, until it is shown by special examination. The vertigo is generally paroxysmal, occurring at intervals of some days or weeks, excited by movements, and by coughing, sneezing, or blowing the nose; or it occurs spontaneously, or even during sleep. The attack sometimes begins with a great increase of the tinnitus, which may resemble the whistle of a locomotive, the firing of a gun, or the roar of a waterfall. The vertigo may be of any of the kinds above mentioned; the patient may appear to turn round, or be thrown forwards, or he may see objects moving round, or to one or other side, or he may himself fall forwards on to one side. The movement, or the sense of movement, both of the patient and the surrounding objects—for these, as a rule, coincide in direction—is generally towards the side of the affected ear. There may be a very short interval of unconsciousness, and, in bad attacks, there is nausea, followed by vomiting, pallor and coldness of the extremities. Sometimes there are movements of the eyes in aural vertigo. The sense of giddiness may last for two or three or several hours, so that the patient is totally unfit for anything, and has to confine himself to the recumbent position; and tinnitus and deafness persist for some time also in an aggravated form.

The attacks in progressive cases may become almost continuous, so that the patient is always suffering more or less from vertigo, and is unable to move at all, while, from time to time, fresh paroxysms increase his sufferings.

Ætiology.—Aural vertigo is rare under thirty years of age: the conditions which may lead to it seem to be cold, gout, syphilis, and senile changes. The attacks are excited not only by movement, but also by fatigue, exhaustion, and gastric derangements.

Pathology.—The exact pathology of labyrinthine vertigo still remains uncertain. By some it is thought that the lesion may be central, occurring in the auditory centres or in the medulla oblongata, and that the symptoms are referred to the ear, as the ocular symptoms of migraine are referred to the eye. On the other hand, it appears certain that the labyrinth is sometimes the seat either of primary disease or of such secondary disturbances of pressure as may result from inflammation of the tympanic cavity. But probably when the lesion is confined to the middle ear, the case should not be termed Menière's disease, and still less if it is in the external ear. The paroxysmal occurrence of the attack in the course of persistent chronic disease has yet to be accounted for.

Vertigo also occurs as part of epilepsy in some cases, and in migraine; it may result from organic disease of the brain, such as tumour. Rarely an *ocular vertigo* results from weakness of one of the muscles of the eyeball; the patient forms a wrong impression as to his surroundings, and the sense of equilibrium is disturbed. *Laryngeal vertigo* (Charcot) is another rare form. The attack begins with burning and crushing pain in the larynx, followed by sharp, dry cough. During the cough vertigo occurs, and the patient may become unconscious, and even have convulsions. A true *gastric vertigo* is much less common than aural vertigo; in many cases supposed to be gastric, the permanent cause lies in the aural apparatus.

Treatment.—Bromide of potassium is of great value in the treatment of aural vertigo; it should be given in doses of 15 to 20 grains three times a day; it probably acts by lessening the conditions of instability of the centre. Blisters or stimulating ointments may be applied behind the ear. Where syphilis or gout is likely to be the cause, it should be met by appropriate remedies, and when the vertigo is associated with conditions of high arterial tension, this should be treated by aperients, and especially by mercury, in the form of blue pill or calomel. Charcot, remembering the effect of large doses of quinine in producing deafness and tinnitus, gave this drug in doses of 7 to 15 grains daily for some months with success. Gowers found sodium salicylate useful in 5-grain doses thrice daily.

In other forms the removal of such causes as can be found, and the use of bromides, are the chief indications. Ocular vertigo is prevented by closing the affected eye.

SPASMODIC TORTICOLLIS.

There are two forms of disease known by the name of torticollis: *fixed torticollis* or congenital wryneck, and *spasmodic torticollis*. The former is due to a permanent shortening of the sternomastoid muscle, which is attributed in some cases to injuries during birth, and is observed first during childhood, if not in early infancy.

Spasmodic torticollis, or spasmodic wryneck, is a functional neurosis, characterised by tonic and clonic contractions of the muscles of the neck, whereby the head is forced into an abnormal position.

Ætiology.—The disease is rarely seen before the age of thirty; it affects both sexes, but females more often than men. The cause cannot always be ascertained; neurotic inheritance, exposure to cold, falls and injuries, overstrain of the shoulder, arm, or neck in particular occupations, have been recorded as antecedents.

Symptoms.—It begins gradually, being first felt as a mere discomfort in the neck; then distinct jerking movements of the affected muscles are felt, by which the head is rotated or displaced. If, for instance, the right sternomastoid is affected, the head is constantly being jerked in the direction of the action of this muscle, the chin is thrust forwards or upwards to the opposite side, and the occiput is drawn down towards the clavicle. The contractions are sudden, irregular, and frequent; for a few moments there is a remission, during which the patient slowly and cautiously tries to bring the head straight, when the muscle again contracts, and the face is gradually forced round to the left. The movements are for a time checked by supporting the head, and they cease during sleep, but immediately the patient awakes the movements recommence, and continue with but little rest throughout the day. The muscle most frequently affected is the sternomastoid, and next to that the upper part of the trapezius and the splenis capitis; the complexus and trachelomastoid, the deep rotators of the head, and the platysma myoides are also sometimes concerned. The position of the head is of course determined by the muscles which contract. The position due to the sternomastoid has been mentioned; the trapezius draws the head backwards and downwards towards its own side, with slight rotation of the face towards the opposite side; while the splenius draws the head downwards with slight rotation towards its own side. Two or more of these muscles may be affected at the same time; most often the sternomastoid with the upper part of the trapezius of the same side, or the splenius of the opposite side. An intermediate position of the head will of course be the result. Or corresponding muscles on the two sides may act together, drawing the head backwards during their contraction. In such cases there is generally an associated contraction of the frontal muscles which normally contract when we throw back the head to look upwards. Occasionally the disease itself spreads to the muscles of the face, or to those of the shoulder or arm; and this is especially so at the height of the paroxysm. In slighter cases, or in early stages, there may be no pain, but in severer forms there is neuralgic pain in the contracting muscles. From their excessive action the muscles often hypertrophy; they at least retain their natural bulk. The electrical irritability is normal, or increased.

Pathology.—Of this little is known. The disease is at least not due to lesion of muscle or of nerve; but is a disturbance of the motor centres, either in the cortex of the brain or in the spinal axis, or perhaps in both situations in the same case.

Diagnosis.—Besides the fixed torticollis above mentioned, distortion of the neck may arise from "stiff neck," a temporary condition of the muscles or fasciæ, and from spasm of the muscles in caries of the cervical vertebræ. Spasmodic wryneck may be simulated by hysteria, but hysteria has often been unjustly assumed when the patient is a real sufferer.

Prognosis is unfavourable; the spasms may subside after a few months, but far more often persist in a more or less severe form for the rest of life.

Treatment.—The disease is most intractable, and numbers of drugs have been tried with but a small amount of success. I have seen good from bromide of potassium. Gowers speaks of benefit from asafœtida and valerianate of zinc. Opiates and other sedatives (chloroform, Indian hemp, morphia) may check the spasms while the patient is under their influence, but it returns again in the course of some hours unless the dose is repeated. Succus conii up to 3 and 4 ounces in a daily dose quiets the spasm for the time only (J. Harley). Galvanism is sometimes useful. A weak constant current should be passed continuously through the contracted muscles for five or ten minutes.

In severe or obstinate cases assistance may be rendered by surgery, and the nerves or the muscles may be divided. If the former are selected, the deep cervical nerves must be resected as well as the spinal accessory. The movements will cease, but a certain amount of incapacity must result from the accompanying paralysis and atrophy: and the disease may reappear in adjacent muscles. Kocher, of Berne, claims success for his method of dividing in two, three, or four operations, the sternomastoid, trapezius, splenius capitis, complexus, trachelo-mastoid, and even the obliquus inferior.

PARALYSIS AGITANS.

This disease consists, in its fully developed form, of a rhythmical contraction of the muscles of the limbs, associated with weakness and rigidity. It has also been called *shaking palsy*, and *Parkinson's Disease*, after the name of him who first described it.

Ætiology.—It is a disease of advanced life, rarely occurring before the age of forty-five years; but, on the other hand, not often commencing after the age of sixty-five. It occurs in men somewhat more frequently than in women. It cannot always be traced to any definite cause; emotion, fright, injuries, acute diseases, and exposure to cold have been the determining factors in some cases.

Symptoms.—It commonly begins in one hand and arm with a tremulous movement, due to a rhythmical contraction of antagonistic muscles. The movement is most marked in the hand; the fingers are generally flexed, with the thumb resting against the forefinger, and the constant slight flexion and extension of the fingers and thumb produces a movement like that required for rolling pills. Similar slight movements of flexion and extension occur at the wrist and elbow joints. After the tremor has existed for some time in one arm, it generally spreads to the leg of the same side; and then in succession to the arm and leg of the opposite side. The trunk may also be affected, though it is not always easy to say how much the tremor is due to the movements in the legs; and, finally, in some cases, there may be a slight movement of the head. Occasionally even the muscles of the jaw and tongue, but very rarely those of the face, are affected. These movements vary in extent; in rapidity they range between 4½ and 7 to the second. As a rule the movements continue even during rest; thus, if the patient sits with the arm resting on the knee, both the leg and the hand and arm will continue to tremble. In early cases, however, support may check the tremor for a time, and in advanced cases with the rigidity to be presently described, the tremor may only occur on movement. By voluntary efforts fixing the limb, the tremor may also for a time be stopped, and it ceases during sleep.

Another feature of the disease is weakness and rigidity of the affected muscles. In the majority of cases this follows the tremor; in others, it occurs at the same time as, or before, the tremor. In a case which has begun with tremor in one limb, the rigidity may appear first in another limb, and be followed by trembling. The muscular weakness is shown by deficient power of grasp, and fatigue on exertion. The rigidity imposes on the patient a characteristic posture, which is most marked when he is standing. The body is bent somewhat forwards, the elbows are flexed nearly to a right angle, and stand out a little from the side, the hands are in the position above described; the legs are slightly bent at the knees; the head is also inclined forwards, and the face wears a fixed anxious expression. The gait is very peculiar; the patient rises from his seat slowly and with apparent difficulty, and his first steps are hesitating; but soon his movements become quicker and quicker, he seems with each step to be trying to prevent a fall, and ultimately, unless stopped, he may actually fall forward to the ground. Some patients when gently pushed backwards are unable to stop themselves, and continue to walk backwards until they meet an obstacle or fall. The terms *festination* and *propulsion* have been used to describe the forward tendency; *retropulsion* and *retrogression*, the backward movement. Speech may be similarly slow at first, and afterwards rapid. Otherwise all movements tend to be slow on account of the rigidity; for instance, the patients turn with difficulty. The muscles are not hypertrophied by their excessive action, and only in late cases with long-continued rigidity do they present some atrophy. The reflexes and electrical reactions are generally normal. Some subjective sensations are often experienced, such as rheumatic pains, sense of fatigue, and especially a sense of great heat, which is often accompanied by free perspirations.

The disease is chronic and progressive, yet its course may be very slow, two or three years perhaps elapsing between the affection of one and another limb. Probably a well-marked case never recovers; but it is fatal only through bed-sores or exhaustion in the extreme cases; intercurrent disease—*e.g.*, of the lungs—terminates others.

Pathology.—The lesion is probably situate in the cerebral cortex, but of its nature nothing is known. Some believe it to be allied to senile degeneration. Recently, sclerotic patches have been described in the spinal cord, starting from the neighbourhood of the vessels (Dana, Redlich).

Diagnosis.—There is but little difficulty in recognising paralysis agitans. Formerly, no doubt, it was confounded with *disseminated sclerosis*, which occurs, however, earlier in life, and in which there are wider and more irregular movements of the limbs brought on by movement only, implication of the head, staccato or scanning speech, and nystagmus. Charcot distinguished *senile tremor* from paralysis agitans, especially by the constant trembling of the head; in the former, also, the movements are finer, both arms are often involved together, and there is no tendency to rigidity: it comes on later in life.

Treatment.—This is most unsatisfactory. Rest and the avoidance of excitement are desirable. Various sedative drugs—morphia, opium, or conium—may check the movements for awhile, but without permanent benefit. Arsenic seems to have done good in some cases, and Gowers recommends arsenic, Indian hemp, and opium together. The continuous galvanic current has occasionally produced slight improvement.

CHOREA.

Chorea (*χορεία*, a dancing) is characterised by irregular involuntary movements of different parts of the body. The popular equivalent, St. Vitus's Dance, has reference to the occurrence in the Middle Ages of epidemics of dancing mania, when patients were cured by a pilgrimage to the shrine of St. Vitus—Chorea Sancti Viti. But the complaint in those epidemics partook rather of the nature of hysteria, and though the name chorea is still sometimes used to indicate some other forms of abnormal movement (chorea major), it is, as a rule, reserved for the disorder now to be described.

Ætiology.—It is mostly a disease of childhood: nearly half the cases occur between the ages of five and ten, and another third between ten and fifteen. It is more frequent in girls than in boys, in the proportion of two or three to one, and it is more common among the poorer classes of society. It is not strongly hereditary in its ordinary form. Among antecedent diseases, acute rheumatism is the most important. About one-third of choreics have had rheumatism: choreic movements sometimes occur in the course of rheumatism, or rheumatic pains during chorea. In some other cases of chorea the attack has been preceded by one of the infectious disorders, such as scarlet fever or measles; or some other septic disorder. Among adult patients, pregnancy is a common antecedent: some of them have had rheumatism, and others chorea in childhood. Fright or mental shock of some kind is certainly a cause of the disease in many cases, though parents are often too ready to account for the attack in this way. It may arise after injury, perhaps also as a result of emotion. The origin of chorea in imitation is probably a very rare event.

Symptoms.—The most prominent feature of the disease is the action of the muscles: they are in a condition of (1) involuntary movement, (2) ataxy or inco-ordination, and (3) slight degree of actual weakness or parexis. The patient is in a constant state of movement, whether lying, sitting, or standing; and the movements, which affect nearly all the muscles of the body, are jerky, irregular, and devoid of purpose. The fingers are opened and shut, the wrist suddenly extended or flexed, or the shoulder lifted. The facial muscles are twitched, the eyebrows suddenly elevated, the head or the eyes rotated to one side, and the chin elevated or depressed. In the lower extremities the movements are often less; the toes are twitched, or one knee gives way. In the muscles of the trunk, one notices half rotation of the body to one or other side, sudden retraction of the abdomen, or jerky action of the respiratory muscles.

The irregularity is more marked on voluntary movements. If the hands are stretched out in front, the child is quite unable to hold them steady; on protruding the tongue, it is put out with a jerk, and perhaps withdrawn suddenly, and the muscles of the jaws act capriciously at the same time; in walking the legs are thrown about, the body is jerked round, and the shoulders are lifted. In the same way it may be seen that the muscles relax with great readiness; after grasping an object, one or two fingers quickly yield, and soon the hand and arm will drop. The movements are increased when the patient is watched, or if she becomes excited; they cease during sleep.

The vocal cords have been seen to quiver, and a low-pitched, monotonous voice is attributed to their want of tension. Speech is irregular, and the patient is unable to sing a long note; these may be due to the irregularity of the respiratory movements.

Sensation is but little disturbed; there may be some formication or tingling, but very rarely any definite hyperæsthesia, or anæsthesia. Both nerves and muscles show increased irritability to faradic and galvanic currents.

It is not always easy to say what the condition of the mind is: often a child with chorea looks silly or idiotic from the purposeless contractions of the facial muscles, which in this case are not a true index of the mind. Apart from this, however, the child's disposition is apt to be altered: she becomes fretful, irritable, capricious, or excited, while intellectually she has a weak memory and is unable to fix the attention.

In about half the cases a murmur over the heart's area may be recognised. The heart is also often irregular in action, but this is probably secondary to the irregularity of the respiratory movements. The murmur is commonly heard at the apex of the heart, and is systolic in time. Mostly it is limited to this area, occasionally it is audible in the axilla and behind, and is obviously due to mitral regurgitation. Sometimes a hæmic basic murmur is present. The origin of the apex murmur has been much discussed, but since endocarditis has been frequently found in fatal cases, and some of the murmurs of chorea are undoubtedly due to a valve lesion, it is fair to suppose that in other cases they often arise from endocarditis. Some, indeed, may be the result of preceding rheumatism; but this will not account for the majority, which appear to develop in the course of the chorea itself.

Varieties.—Sometimes the symptoms are very slight, and remain so for some time; the fingers are only twitched a little, irregular movements are scarcely noticed, but the child drops things that she attempts to carry.

In some cases the movements are limited to the arm and leg of one side only (*hemichorea*); but the facial and trunk muscles are always involved on both sides.

A decided paralysis is sometimes present together with slight choreic movements; the arm hangs by the side, and can with difficulty be raised; the fingers are twitched occasionally, and the grasp is extremely feeble (*paralytic chorea*).

Exceptionally the movements are very violent; standing or sitting is impossible, and the patient is confined to bed, where she throws herself about in the wildest contortions, striking the hands and arms against the sides or head of the bed, and rubbing the elbows, shoulders, buttocks, hips, knees, and heels, so as to produce serious abrasions of the skin. Feeding becomes difficult or impossible, as everything placed to the mouth of the patient is jerked aside, or spilt; and even if it gets into the mouth it may be rejected by the want of co-ordination for deglutition. These cases (*chorea gravis*) sometimes progress with great rapidity; the patient is exhausted by the constant movement and the want of sufficient nutriment; rapid emaciation takes place, the face is flushed, the eyes sunken but bright, the lips and tongue dry, the pulse rapid, and ultimately death may occur, being preceded often by some rise of temperature. In some the mind is severely affected, and the patient becoming delirious, or even wildly maniacal. Such violent cases are much more frequent in adults between the ages of fifteen and twenty-five, and a large proportion are in pregnant females.

Duration.—The duration of chorea is very variable. The majority of cases last from six weeks to three months; not infrequently some slight twitching may occur for many weeks or months after the severer manifestations have subsided, and the symptoms may again after a time become aggravated. In the end most cases recover. The violent cases are usually of short duration; if death takes place it is often within two or three weeks from the first symptom, or from the time when the movements became violent; if recovery ensues the movements become quieter after a few weeks, though complete cure may be delayed some time. Chorea is very apt to recur even after its entire subsidence; second and third attacks are frequent. These may be of shorter duration than the primary attack, but are not different in other respects.

Sequelæ.—The disease sometimes leaves behind it a liability to sudden starts, which in the course of months subside. In some cases towards the end of the attack, paralysis of the limbs occurs. This may be only on one side (*choreic hemiplegia*); but rarely all four limbs are affected, the child lying quite helpless, and each limb dropping like a log on being raised from the bed. Speechlessness, mental weakness, maniacal and melancholic conditions, also occasionally occur, and are generally temporary. Epilepsy has also been observed as a sequela of chorea. The endocarditis may terminate in chronic valvular disease.

Morbid Anatomy.—The nervous system presents after death no appearances, either to the naked eye or microscopically, that can be regarded as essential to the disease, or in any sense its cause. Such changes as have been found vary in different cases, and are either accidental or secondary. Among these may be noted obstructions of minute vessels in the brain, swelling and degeneration of nerve-cells in the corpora striata and other parts, enlargements of the perivascular spaces, and hæmorrhage around minute vessels. Of the other organs of the body the heart is the only one that is generally involved; and in fatal cases of chorea this nearly always presents evidence of endocarditis (75 out of 80 cases, Sturges; 17 out of 18 cases, Fagge). Fine granulations are found along the edge of the mitral valve on the auricular face, and sometimes on the aortic valves. These are present even when there has been no antecedent rheumatism. Occasionally the valvular lesion is older and more extensive.

Pathology.—The seat of the lesion and its nature have to be considered. Many of the facts of chorea are in favour of the disturbance being cerebral, and involving chiefly the motor convolutions. They are—its origin in emotional disturbance, the influence of the will, of emotion, and of distraction of the attention upon the movements, their cessation during sleep, their frequent limitation to one side in the limbs, while they affect both sides of the face and trunk, and the coincident disturbance of the mental faculties. But so far no corresponding structural changes have been found. It is highly probable that chorea will be proved some day to have a toxic or microbic origin. The facts in favour of this are:—The frequent occurrence of endocarditis, and its almost universal presence in the fatal cases; the association with rheumatic fever, and other diseases more certainly infectious; and the mode of death, which is by no means always explained by simple muscular exhaustion. Chorea would be thus analogous to tetanus and hydrophobia, in which muscular movements are determined by the circulation of poisons, without the presence of demonstrable nerve-lesions to account for the symptoms.

Diagnosis.—This rarely presents any difficulty. Movements closely resembling those of chorea may occur as a part of *hysteria;* they are generally more rhythmical, more localised, and may recover quickly. The jerky movements of Friedreich's ataxia may cause confusion; but in this the gait is different, the history is a very long one, and nystagmus is present.

Prognosis.—In children it is favourable, apart from the condition of the heart; in young adults it is much more uncertain.

Treatment.—The child should be kept quiet in bed, and everything tending to worry or annoy should be kept from her. She should not be subject to the ridicule of companions, nor to much study of lessons. The diet should be plain, nutritious, and abundant. The drugs that have been most useful are zinc and arsenic. The latter undoubtedly shortens the attack; it may be given in 2 or 3 minim doses of liquor arsenicalis three times a day after meals, and gradually increased to 5 or 7 minims in young children, or to 10 minims in those who are approaching puberty. Sulphate of zinc is less certain; it is given in doses of 2 grains three times a day, increased every two or three days by additions of 1 grain to each dose up to 10 grains; thus increased gradually it does not generally produce sickness. The salts of iron are also sometimes given. Bromide of potassium and hydrobromic acid have been used, but without much success. Antipyrin in doses of 5 to 7 grains appears to be of use sometimes. In severe cases in children I have found, with Goodhart, that massage is of benefit in reducing the disease to more moderate limits. In the very violent cases treatment becomes difficult. The patient must be protected from injury by padded boards at the side of the bed, the nutrition must be maintained, and food may have to be given per rectum. Chloral is probably the best drug to administer, but must be given with caution; morphia is less desirable. Quiet may be obtained for a time by inhalations of chloroform, but the movements return as the effect passes off. Conium and Calabar bean may also be tried. In cases of paralysis after chorea I have seen good results from strychnia. When the movements are slight in mild cases, or in recovering stages, gymnastic exercises or the skipping-rope may be found useful.

CHOREA IN ADULTS.

Besides the young adults who suffer from the chorea already described, persons of middle and advanced age are affected sometimes with a similar disease. The movements are generally more extensive, often prevent the individual from following any occupation, and may persist for years; though some cases are of quite short duration and recover. These cases seem to have no connection with rheumatism or cardiac disease, but may arise from fright or severe emotion.

Under the name of *Huntington's Chorea* is described a form which occurs in several members of the same family, affecting males more than females: it appears late in life, and is associated with definite insanity.

TETANY.

In this complaint there are peculiar contractions of the muscles of the hands and feet occurring in paroxysms.

Ætiology.—It occurs at all ages, but is especially frequent in infants and in young adults. In children, males are more often attacked than females; in older people the reverse holds good. In children, rickets and diarrhœa are predisposing causes; in women, pregnancy and lactation; and in adults of both sexes exposure to cold, recovery from febrile diseases, and the existence of dilatation of the stomach. It has occurred after the operative removal of the thyroid body: and similar paroxysms have been seen as the result of ergotism.

Symptoms.—There may be some preceding discomfort or malaise, or stiffness in the arms, or tingling for some hours or days. Sometimes the paroxysm comes on suddenly without warning. The hands are then bent on the wrist, the fingers are flexed at the metacarpo-phalangeal joints, extended at the phalangeal joints, and pressed closely together with the thumbs bent into the palm of the hands; so that the hand assumes a conical shape. The elbows are slightly flexed, and the arms adducted to the sides. In the lower extremities, the foot is extended on the leg, the tarsus arched, and the toes flexed and crowded together. These are the characteristic contractions, and in most cases these alone occur. In very severe cases spasm affects the muscles of the abdomen, chest, face, and tongue, as well as those of the back, causing slight opisthotonus, and of the eyes, causing strabismus. There may be some cramp-like pain in the parts affected; the back of the hands may be tumid, and the veins swollen. There may be sweating, flushing, and slight rise of temperature. The spasm ceases in from five to fifteen minutes, or it may last one, two, or more hours; it gradually subsides, and recurs again after an interval of some hours or days.

In the intervals the nerves and muscles show an increased susceptibility to mechanical irritation. Percussion of the nerves causes contractions in the corresponding muscles, and stroking the face from above downwards causes contraction of the muscles one after the other. Trousseau first showed that in the intervals a fresh paroxysm could be brought on in a few minutes by firmly grasping the arms, or by pressure on the nerves and arteries. The nerves also are unduly susceptible to both faradism and galvanism. Very weak galvanic currents applied to them cause contraction of the muscles. ACC is most readily obtained, and both anodal and kathodal contractions are of long duration. But though an interval between the paroxysms is here spoken of, it does not always occur. In infants a continuous spasm is more common, and in adults the spasm may not entirely relax; so that this form is called *remittent*, while the form with complete intervals is called *intermittent*.

The disease lasts from a few days to a few weeks, and recovery is the rule. But death may occur from exhaustion when the paroxysms are severe; or from pneumonia as a result of interference with respiration; or in infants from the diarrhœa which caused the disease. Occasionally, some weakness of the legs remains for a short time after recovery; and muscular atrophy and fibrillary tremors have been seen.

Pathology.—*Post-mortem* examination has not revealed the cause of the disease. Its associations suggest a toxic origin; its distribution and the implication, though slight, of the sensory, nutritive, and electrical functions indicate that the peripheral nerves may be the parts undergoing irritation (Judson Bury).

Diagnosis—The distribution of the spasms—namely, their occurrence chiefly in the hands and arms—distinguishes it from *tetanus*. *Hysterical* contractions may take on the form of tetany;

they are generally unilateral, and are associated with other hysterical conditions.

Treatment.—Bromide of potassium should be given in full doses, and the predisposing condition of the patient should be as far as possible removed. Thus, in children, diarrhœa should be treated, and rickets met by cod-liver oil, iron, suitable diet, &c. Women should give up nursing their children, and should take iron and other tonics. Other drugs—namely, chloral, Indian hemp, Calabar bean, and morphia—have also been given with more or less success. Chloroform inhalation stops the spasms for a while. Galvanism with the anode on the nerves may also be tried.

HYSTERIA.

By hysteria is meant a disorder of the nervous system, which leads to various functional disturbances, sensory, motor, or visceral, of varying duration, and almost certainly curable. The disturbances may be numbness or pain, paralysis, spasm, or general convulsions, flushes, palpitation, retention of the urine, and numerous other allied conditions. They occur from time to time in the same individual, and are generally spoken of as manifestations of hysteria, or hysterical symptoms or attacks; while the name hysteria is rather reserved for the general condition of the nervous system which is the cause of these disturbances, or forms the tendency to their occurrence. A person who has once had an attack, or a symptom of the kind called hysterical, is believed to have a tendency to further attacks, and practically to be one affected with hysteria. It is, in great part, a disorder of the higher functions of the brain—viz., the mental faculties, and it has been variously regarded as due to a predominance of the emotional faculty, or a subordination of the will to ideas or emotions rather than to the intellect or judgment. But such conceptions do not cover the whole of the manifestations of hysteria, especially the visceral or vasomotor disturbances. In so far as the will seems to be concerned in any particular symptom it is important to understand clearly that hysteria is not *shamming* or *malingering*, though it may resemble it closely, and though in any given patient there may be a gradual transition from the condition of a genuine hysteric to that of a wilful deceiver. The patient with hysterical paralysis, or spasm, is not wilfully representing herself as unable to move the leg when she really can; she is at the time totally unable to supply the voluntary power necessary to move the limb, and if a sudden recovery should take place from excitement, the action of a galvanic battery, the threat of some disagreeable proceeding, or, what is more common, if she should slowly recover, from the steady moral pressure of encouragement and the absence of unnecessary sympathy, this is because the will is suddenly or gradually stirred up to exertion by fresh motives or impressions upon the judgment or emotions. It is, indeed, not so much the will to do wrong as the absence of will to do right. Sensory manifestations can be referred to a perversion of the will only by assuming that the patient wills to falsely represent herself as feeling pain, or as having lost common sensation; and some of the visceral disturbances are of such a nature as apparently to be entirely beyond direct voluntary control.

It is necessary to point out that the name hysteria (ὑστέρα, the womb) was given with the idea that a disorder of the uterus was the essence of the disease; indeed, it was even believed that the womb moved about from place to place in the body, and caused the various symptoms that will presently be described. But it is now known that although sexual relations may have much to do with many cases of hysteria, this is not invariably the case, even in female adults; while the disorder occurs frequently in quite young girls, in boys, and occasionally in men. It is likely, however, that the name will be long retained, as it sufficiently indicates a disorder which is protean in its variety, the exact pathology of which still remains more or less of a puzzle, and the limits of which are not with accuracy defined.

Ætiology.—Hysteria affects chiefly females between the ages of fifteen and fifty; but it occurs in older women, and not infrequently in quite young girls. Adult males rarely, but boys more often, become subjects of the disease. Heredity has an important influence; hysteria is likely to appear in the offspring of hysterical parents, as well as in the children of the insane, of habitual drunkards, and of the sufferers from other neuroses. The hysterical tendency is further fostered by bad moral training in the child, such as allows it to yield to every emotional impulse, makes it expectant of undue sympathy in all its slight complaints, and does not teach it to be independent in battling with the troubles of everyday life.

Hysterical attacks are brought on by mental and physical disturbances of all kinds. The most frequent are emotional: domestic or business anxieties, grief for the loss of friends or relatives, serious quarrels or a mere difference of opinion, arrival at a rough part in the course of true love, are often the occasions of hysterical outbursts. Among physical causes we may have direct injury; thus, a blow on the stomach may cause gastralgia and flatulence, or an injury to the arm may be followed by hysterical paralysis or spasm. General illnesses, by their weakening effect, may cause various hysterical symptoms; or local lesions may lead to hysterical manifestations of the part primarily diseased. Thus a faucial or laryngeal catarrh may be followed by hysterical aphonia, or an actual synovitis by hysterical pain in the joint. Among the local causes of hysteria may be mentioned diseases of the uterus—especially flexions of various kinds—and disease of the ovaries, viz., ovaritis. The former may be influential by acting through the emotions, perhaps to a greater degree than other local lesions; but the cure of the malposition by no means always cures the hysteria. As to the ovaries, it has been observed that in many cases of hysteria there is tenderness on deep pressure in the iliac region, and this has been ascribed to a tender ovary; but not always, it would seem, with sufficient grounds. Firm pressure at the same spot will also sometimes put a stop to a violent hysterical attack.

The symptoms of hysteria may be described as mental, sensory, motor, vasomotor, and visceral.

Mental Condition of Hysteria.—As already stated the subjects of hysteria generally show a deficiency of will with an excessive development of the emotional faculties. They give way readily to grief, or joy, and seem to have little power of controlling the flow of tears, or the outburst of laughter. They are self-conscious, and show a constant desire for the sympathy of those around them. Thence arises a willingness to continue ill for the sympathy it excites, and in extreme cases the artificial production of symptoms and lesions by which their friends, and even their medical attendants, are led to believe them to be seriously ill. For instance, a girl will produce an artificial eruption on the skin by nitric acid, or cantharides powder, or the ends of matches; or will make a tumour in the face by accumulating substances between the gum and the cheek; or will show some foreign body that she states has formed in the rectum or vagina. Such performances one can scarcely doubt are intentional, but they are connected by every gradation with the quite involuntary simulation of disease, and occur, as a rule, in those who for other reasons may properly be regarded as hysterical.

Sensory Symptoms.—Of these may be noticed increased acuteness of the senses, amounting to intolerance of light or sound, or extreme hyperæsthesia. The patient will insist on the blinds being drawn, and complain of the least noise, or the slightest touch or shock. Tenderness is often noted at different parts of the body, especially the spine, the ovaries (as already mentioned), the left hypochondrium, the infra-mammary region, or the top of the head. Sometimes pressure in one or other of these regions causes pain radiating from the spot, followed by *globus* (*see* p. 354), and even by a complete convulsive fit. Such spots have been called *hysterogenic*. Numbness and tingling, or other dysæsthesiæ, are occasionally felt. Sometimes sensation is lost in one or other limb, or in one half of the body, constituting *hysterical hemi-*

anæsthesia. The loss of sensation is then strictly limited to one half of the body, ceasing sharply at the middle line, and involving the mucous membranes as well as the skin; the patient is insensitive to pain, so that pins may be thrust deeply into the skin without her knowledge. Hemianæsthesia is accompanied by defects of the special senses of sight, hearing, smell, and taste on the same side of the body. The affection of sight is a crossed amblyopia; it involves the sharpness as well as the field of vision, and the fields of the several colours (*see* p. 185). Sensation has been sometimes restored to a part by the local application of metal plates, especially iron, copper, zinc, or gold (*metallo-therapy*), and under these circumstances anæsthesia may be found at the corresponding spot on the previous healthy opposite side of the body. This so-called *transfer* can be brought about in the case of other hysterical symptoms, such as paralysis and contractions, and by other agents, such as large magnets, feeble galvanic currents, static electricity, vibrating tuning forks, or sinapisms. It is no doubt a mental process.

Motor Symptoms.—*Paralysis.*—Hysterical aphonia is not uncommon; it results from paralysis of the adductors of the vocal cords (*see* Paralysis of the Laryngeal Muscles). It is important to note that adductor paralysis, without abductor paralysis, is always a functional disorder. On the other hand, abductor paralysis, existing alone, is mostly due to organic disease, and very rarely the result of hysteria; it causes stridor and dyspnœa, and may even in hysteria lead to dangerous asphyxia. Dysphagia may arise from functional paralysis of the pharyngeal muscles. Ptosis also occurs as a hysterical symptom; it may be single or double. Paralysis of the limbs occurs in the form of paraplegia or hemiplegia, or all the limbs may be paralysed together. The paralysis in these cases is often not complete, and if the patient makes an effort to move the limb in a particular direction, it may be seen that some antagonistic muscles contract. The patient may assert that she is unable to lift the arm, yet if it is raised by any one else she will often keep it supported or let it drop only half-way, showing that the muscles believed to be paralysed have still a considerable amount of power. Also, if the attention is distracted to other things the patient may unconsciously move the supposed useless limb. If one lower extremity is alone affected, on attempting to walk the patient drags the paralysed limb behind her, making no effort to bring it forward, but only hopping along on the sound limb. The nutrition of the muscles and the electrical reactions are normal. Knee-jerks are generally normal, and there is no true continuous ankle-clonus, but there is often a clonus lasting only a few seconds, and in some cases the knee-jerks are excessive. In paraplegia the legs can often be moved in bed, but the patient is quite unable to stand, and there is never incontinence of urine or fæces; in hemiplegia the leg is sometimes worse than the arm, and the face and tongue are always spared. Paralysis is sometimes, but not always, accompanied by anæsthesia. In a rare form of hysteria, every attempt to move, or contract a muscle, is painful (*akinesia algera*). Another form of hysteria is a disorder called *astasia-abasia*, in which the patient can neither stand nor walk, though he can move the legs in bed, and there is neither inco-ordination nor sensory failure.

Ataxia. — Occasionally a well-marked ataxia is present in hysteria, either alone, or associated with paralysis.

Tonic Contractions.—A common manifestation of hysteria is the tonic contraction of one or more muscles, often for long periods of time. Such contractions may come on after recovery from hysterical fits, and they may be excited directly by a blow or by emotional disturbance. The arm or the leg, or both together, are sometimes affected; the arm is generally flexed at the elbow, and drawn close to the side; the leg is rigidly extended. The limbs resist any effort to change their position, and the muscular contraction is stronger the greater the external force applied. It does not relax even in sleep, but only in the deepest chloroform narcosis. Rigidity of both legs is not common; when it occurs it is mostly in the position of extension. Contraction of all four limbs is very rare. Trismus, or closure of the jaw, is another form of hysterical contraction. Anæsthesia may be present at the same time.

Contractions may last months or years, or they may suddenly cease under the influence of emotion, or of the application of the faradic current.

Clonic Contractions.—These may occur in the form of tremor, or of more decided rhythmical movements. The latter may be nodding or rotatory movements of the head, or spasms of the muscles at the back of the neck, producing a spurious torticollis (Gowers); or one fist may be constantly beaten upon the opposite hand, or the knee or thigh. Some movements have a rather close resemblance to ordinary chorea, and have been called hysterical chorea; they are generally more sudden, more regular and rhythmical, and less like combined movements. The extensive movements of the whole trunk in bowing and swinging, which have been described as chorea major, are mostly of a hysterical nature.

The Hysterical Fit.—The attacks known as hysterical convulsions, and "fits of hysterics," are commonly caused by emotional disturbance. Nevertheless they may occur in the middle of the night. An attack begins with *globus hystericus*, a sensation as if a ball were rising in the throat, threatening to choke the sufferer; with this there is giddiness, or palpitation, and the patient may burst into a fit of crying, or of uncontrollable laughter. In other twitch

cases after the globus the patient falls to the ground, or on to a chair or sofa, and at once passes into convulsions. These may at first be of a tonic kind; the body and legs are rigidly extended; the body arched forwards in a state of opisthotonus, with perhaps only the head and heels touching the ground; the arms are rigidly extended, either close to the body, or at right angles to it, and the hands are clenched. The movements that succeed are of the most varied description; often they have every appearance of being made with a purpose. The back of the head may be repeatedly dashed against the floor until it actually bleeds; the limbs are thrown wildly about, and the bystanders are struck, or clutched at; if the limbs are restrained the struggling and fighting become more violent. Sometimes the patient gnashes with the teeth, and may groan or shriek. The eyelids are generally closed, and resist attempts to open them; if they are opened, the eyeballs are rolled upwards under the upper lid. The face is usually red, and not livid as in epilepsy. There may be some saliva issuing from the mouth, but the tongue is not, as a rule, bitten. Consciousness is not entirely lost; the patient does not answer questions, but her actions may be guided by what is said in her presence, and, as already stated, there is automatic resistance to those who restrain her. After the active struggling movements have continued some minutes they commonly cease, and the patient lies panting, with closed eyes, muttering, or delirious, not responding to the appeals of her friends, until she again goes off into convulsions. These alternations may be repeated for two or three hours. Recovery is often quite rapid; the movements cease, the patient opens her eyes, and looks round, wonders what she has been doing, or, recognising it from former experience, may burst out crying. Headache may be present for some time afterwards, and a recurrence of the attack is not infrequent within a few days. The patients state that they have no knowledge of what has happened.

French writers have made us acquainted with a severe form of hysterical attack (*hystero-epilepsy*, *hysteria major*) which is not often seen in England. The attack is preceded by a *prodromal* period, in which there are changes of manner, generally in the direction of melancholy, with hallucinations of sight and sound; disturbances of the organic functions, such as nausea, indigestion, borborygmi, hiccough, yawning, palpitation, &c.; muscular weakness, unsteadiness, or twitchings; and finally, sensory troubles, such as anæsthesia or hyperæsthesia. Ovarian hyperæsthesia is a marked feature, and the *hysterogenic spots* (or *zones*) above described (p. 352) are found especially in the following regions:—Supra-mammary, mammary, infra-mammary, infra-axillary, hypochondriac, iliac, ovarian, upper dorsal, and lower dorsal. The attack itself is divided into four periods: (1) The epileptoid period,

twitch

the periods (2) of contortions and great movements, (3) of
tional attitudes, and (4) of delirium. In the first period there
tonic and clonic stages, as in epilepsy, with unconsciousness ; t
last from two to five minutes, and are followed by resolut
The most striking feature of the second period is an extreme f
of opisthotonus, the body resting for a moment on the soles of
feet and the vertex, and then dropping flat on to the bed. T
movements are frequently repeated. Violent flexions and ex
sions of the body and other movements also take place.
period of attitudes follows upon this: the patient is obvio
subject to vivid hallucinations, and assumes in succession attit
of grief, joy, terror, erotism, &c., in accordance with the lea
idea. The "position of crucifixion" (extension of the trunk
legs with the arms at right angles to the body) is often associ
with a subdued expression of happiness. The patient then pa
into a condition of delirium, of the most varied kinds, and in
end gradually recovers.

Visceral Symptoms.—Globus hystericus has been alre
mentioned, and dysphagia ; the latter may be due to spasm
the œsophagus, which generally yields to steady pressure
the bougie, if this should be required to distinguish it f
organic stricture. Vomiting, gastralgia, and flatulent disten
are common accompaniments of hysteria. Anorexia may
marked feature, and food may be refused for long periods ; o
in these cases some deception is practised, and food is ta
secretly. The so-called fasting-girls are usually hysterical i
viduals whose ailments are fostered by the sympathy
ignorant wonder of the public, and the love of gain of t
immediate friends. The *anorexia nervosa* of Sir William Gu
a condition allied to hysteria, in which the patient may be red
to an extreme degree of emaciation by the continued refusa
food On the side of the circulation we may have palpitat
flushings, rapid or slow pulse, cardiac pain or pain like ang
The respiration may be extraordinarily rapid, and reach 70,
or 90 in the minute without any pulmonary lesion ; the pat
is able to go about without much distress; and during sleep
respirations fall to 20 or 18. Hysterical cough is common ;
generally incessant and noisy, or "barking" in character.
stant hiccough lasting for hours or days may be a hyste
feature. The urine passed after a hysterical fit is generally a
dant, pale, and of low specific gravity. A diminution of secre
ischuria, occasionally occurs. Retention of urine is not
common in hysteria, incontinence is rare ; it is often said
hysterical women never wet their beds. Similarly, constip
occurs, but never incontinence of fæces, and rarely diarr
Elevations of temperature to 110°, 116°, and even 122° Fahr.
been recorded from time to time, which can only be expla

on the view that they are related in some way to hysteria. They may occur in patients ill of other diseases, such as phthisis, and may be sometimes the result of compression of the bulb of the thermometer, or the application to it of hot flannels or poultices on the part of the patient. The temperature is rarely the same in different parts of the body; or at different times within the same hour. But a true *hysterical pyrexia* undoubtedly occurs, either in the form of a fever continuous over some days, or as slight occasional rises to 102°, 103°, or 104°.

Some other conditions occur in hysterical subjects, which have often been described separately. In *catalepsy* the limbs tend to remain for long periods in whatever position the observer likes to place them. The patient appears to be deprived of voluntary movement; when another tries to lift the limb there is at first some resistance, then it yields, and if placed in a new position it remains so for a long time; ultimately, however, the limb will yield to gravity, and gradually sink into a position in accordance with it. The peculiar nature of the resistance to passive motions has led to the term "flexibilitas cerea." Catalepsy is often accompanied in hysteria with disorders of sensations. Besides forming a part of hysteria, catalepsy may occur after weakening illnesses in those who have shown no other indications of hysteria, in some mental affections, and occasionally in meningitis and apoplectic coma. It may also form a part of induced hypnotism.

Trance or *lethargy* is, like catalepsy, sometimes the result of hysteria, or of exhausting illness, or of hypnotism. The patient is in a peculiar condition resembling sleep, and may remain so for days or even weeks. The face is pale, the limbs relaxed, the eyelids resist efforts to open them. The pupils are moderately contracted or dilated, and react to light. The pulse is small, the heart-sounds are feeble or inaudible, and the breathing is extremely quiet, so that occasionally the patient has been thought to be dead. In prolonged cases there are remissions in which the patient may take food, relapsing again into stupor. Most cases recover. *Somnambulism* is another development of hysteria.

Hysteria has some close relations with insanity; many insane women have previously been hysterical, and it is not always easy to draw the line between the two states.

Diagnosis.—This depends chiefly on the age and the sex, which have been already specified; on the fact of previous manifestations; and on the history of the commencement of the present complaint, which is often sudden or occurs after a fit or other hysterical symptom, or after an emotional disturbance or blow quite inadequate for the production of an organic lesion. The absence of organic disease is in favour of, but its presence does not disprove hysteria. Hysterical disorders are variable in degree from day to day, and are often less pronounced when the attention is distracted to other things. Further, the patient may suffer from different symptoms successively, the first recovering as the later ones appear. Some disorders are in themselves characteristic, such as—globus hystericus, aphonia from adductor paralysis of the larynx, and hemianæsthesia. The distinctive points about the paralyses are, especially, the variability from day to day, the unconscious contractions of antagonistic muscles when the attempt is made to move a limb, and of the muscles supposed to be paralysed when the patient's attention is withdrawn. The diagnosis of hysterical from epileptic fits has already been discussed (*see* p. 326). But true epileptic fits are sometimes succeeded by hysteroid convulsions.

Prognosis.—The sufferer from hysteria almost invariably recovers from her symptoms or attacks, though it may be after a very long time in cases where their nature is not recognised; and with advancing age the disease itself dies out. The possibilities in the other direction are that death might occur from exhaustion of hysterical vomiting, or anorexia; or from asphyxia in abductor paralysis. Charcot records a case where a prolonged hysterical contracture was followed by lateral sclerosis of the spinal cord.

Treatment.—Both the general tendency to hysteria, and the particular form it takes in the case before one, have to be considered; and in either case the treatment may be both moral and physical. The patient should be placed under the best hygienic conditions. Fresh air, good food, moderate but not exhausting exercise, relief from mental overstrain or worry, the use of blood-tonics such as iron, and the condition of the bowels, should all be considered. In the pursuance of moral treatment, it may be necessary to remove the patient from her friends, whose ignorant sympathy only prolongs the morbid condition. Many patients recover at once in hospital, where the sympathy they get is no more than in proportion to the danger of their case. In some extreme cases, after milder measures have failed, success has attended the use of the Weir-Mitchell method, in which the patient is not only removed from her friends, but is absolutely isolated, and visited only by her nurses and the physician, until some improvement has been made. She is at the same time kept entirely at rest, fed abundantly, and submitted to the operations of massage and faradism. The former process directly favours the flow of lymph and venous blood in their respective vessels, and the latter, by contracting the muscles, improves their nutrition, and assists the effect of massage upon the vessels. The moral influence of the physician should be in the direction of encouragement to do things that appear impossible, of promise that recovery will come with patience and perseverance, of no undue sympathy for her in her ailments, even the reverse of sympathy in some of on the view that they are related in some way to hysteria. They may occur in patients ill of other diseases, such as phthisis, and may be sometimes the result of compression of the bulb of the thermometer, or the application to it of hot flannels or poultices on the part of the patient. The temperature is rarely the same in different parts of the body; or at different times within the same hour. But a true *hysterical pyrexia* undoubtedly occurs, either in the form of a fever continuous over some days, or as slight occasional rises to 102°, 103°, or 104°.

Some other conditions occur in hysterical subjects, which have often been described separately. In *catalepsy* the limbs tend to remain for long periods in whatever position the observer likes to place them. The patient appears to be deprived of voluntary movement; when another tries to lift the limb there is at first some resistance, then it yields, and if placed in a new position it remains so for a long time; ultimately, however, the limb will yield to gravity, and gradually sink into a position in accordance with it. The peculiar nature of the resistance to passive motions has led to the term "flexibilitas cerea." Catalepsy is often accompanied in hysteria with disorders of sensations. Besides forming a part of hysteria, catalepsy may occur after weakening illnesses in those who have shown no other indications of hysteria, in some mental affections, and occasionally in meningitis and apoplectic coma. It may also form a part of induced hypnotism.

Trance or *lethargy* is, like catalepsy, sometimes the result of hysteria, or of exhausting illness, or of hypnotism. The patient is in a peculiar condition resembling sleep, and may remain so for days or even weeks. The face is pale, the limbs relaxed, the eyelids resist efforts to open them. The pupils are moderately contracted or dilated, and react to light. The pulse is small, the heart-sounds are feeble or inaudible, and the breathing is extremely quiet, so that occasionally the patient has been thought to be dead. In prolonged cases there are remissions in which the patient may take food, relapsing again into stupor. Most cases recover. *Somnambulism* is another development of hysteria.

Hysteria has some close relations with insanity; many insane women have previously been hysterical, and it is not always easy to draw the line between the two states.

Diagnosis.—This depends chiefly on the age and the sex, which have been already specified; on the fact of previous manifestations; and on the history of the commencement of the present complaint, which is often sudden or occurs after a fit or other hysterical symptom, or after an emotional disturbance or blow quite inadequate for the production of an organic lesion. The absence of organic disease is in favour of, but its presence does not disprove hysteria. Hysterical disorders are variable in degree

Weir-Mitchell treatment; in anorexia the feeding must be abundant and frequent; in vomiting, food may be supplied by the rectum, and by the nasal tube. The discomfort caused by both of these methods no doubt contributes to their success.

NEURASTHENIA.

This term is now in frequent use to designate conditions which mostly result from exhaustion of nervous power. These may arise in men as well as in women, and do so especially in those who have been submitted to prolonged mental and physical strain, business anxieties, &c. Such persons find themselves after a time unequal to any physical or mental exertion, and suffer from various symptoms, such as oppression at the top of head, frontal or occipital headache, weakness of the eyes, loss of sleep, and giddiness; loss of appetite, indigestion, constipation of the bowels, with sallowness and some loss of flesh, deficient cardiac power, and hence cold hands and feet; or it may be pain in the back, with tenderness at one particular vertebral spine (*spinal irritation*), pain radiating down the limbs, numbness, or tingling, &c. These, as will be seen, are so many functional disturbances that may be induced, some in one case, some in another, by a want of proportion between bodily income and expenditure: they scarcely constitute a separate disease. Those who suffer from these troubles are not necessarily hysterical; indeed, they present some important differences. Hysteria occurs almost exclusively in women: neurasthenia is seen often in men. Hysteria occurs in those who have but little to do: neurasthenia in those who are overworked mentally or physically. The hysterical woman looks for sympathy: the patient whose nerve-force is exhausted often conceals his deficiencies and strives to overcome them.

Treatment.—Rest is the chief, as it is obviously the most natural, treatment; and it must be both bodily and mental. Good and abundant food must be given, and the bowels regulated; stimulants, narcotics, and anodynes as a rule should be avoided. Recovery is often assisted by the Weir-Mitchell method (p. 358); but massage of itself may be a useful adjunct to bodily rest, without the moral advantage of isolation. The treatment may be supplemented by change of air and scene.

MASSAGE.

It may be well here to say a few words upon a means of treating numerous ailments, which has proved of much value, although there is a tendency, no doubt, to expect success from it in far too many diseases. *Massage* is the systematic manipulation of the limbs and other parts of the body. In the cases in which its use has been advised, it is mostly the muscles that are manipulated, as, for instance, in paralysis, contractures, hysteria, neurasthenia, &c. Several distinct methods of manipulation are comprised under the term. One is *effleurage;* the part is stroked with the palm of the hand in an upward or centripetal direction, and the hands are used one after the other with regularity, and more or less quickly according to circumstances. In *pétrissage* a portion of muscle is picked up with the fingers and thumb of one or both hands; it is subjected to firm pressure and rolled between the fingers and the subjacent tissues. Successive portions are taken up one after another, working, as in the previous exercise, from periphery to centre. *Friction* is rubbing the surface of the limb with the tips of the fingers. *Tapotement* is percussion with the tips of the fingers, the palmar surfaces of the tips, the palms of the hand, the back of the half-closed hand, the ulnar or radial border of the hand, or the whole hand hollowed so as to enclose some air between it and the surface of the limb.

All the movements should be centripetal; they should be done with the dry hand, without the intervention of oil, ointments, or liniments. The duration of massage should be from five to fifteen minutes on any one occasion; but in recent cases the sittings may be three or four in the day. The effect of these manipulations is to promote the flow of lymph and blood in their respective vessels, and to stimulate the muscles of the skin and the skin reflexes.

HYPOCHONDRIASIS.

This is really a mental disorder, which in its mildest forms comes frequently under the notice of the general physician, and only in extreme cases requires the same special care as is given to the insane. It consists in a morbid anxiety on the part of the patient as to his own health, and a morbid magnification of his sensations, so that he imagines illnesses that do not really exist. It is more common in men than in women, and most frequent between the ages of twenty and forty. There is sometimes a hereditary taint of insanity from the parents; and the complaint may be originated by various depressing circumstances, such as business anxieties, moral considerations, the existence of gout, or slight digestive disturbances.

The sufferer from hypochondriasis is constantly under the impression that he is the subject of serious disease; every sensation that he has contributes to this idea, and he can turn his attention seriously to nothing else. He scrutinises with the utmost care his tongue, the colour of his skin, the consistence and colour of his motions, and magnifies every abdominal sensation into a wearing or acute pain, which must be, according to him, due to cancer, or to internal ulcer, or to some other serious disease of which he has heard; whereas a most thorough examination fails to reveal anything at all, or at most some trifling disturbance of the stomach or bowels. In a large number of the cases the complaints have reference to the abdomen; but there are other patients whose only concern is their sexual functions. They are frequently unmarried men between the ages of twenty and thirty-five, who have mostly led chaste lives, but may have masturbated when younger. Occasional pollutions at night, and the escape of a little prostatic secretion after defæcation, lead them to believe that their "vital fluids" are draining away from them; they complain of weakness, giddiness, oppression on the top of the head, inability to attend to their business, loss of memory, and shyness in presence of the other sex; they are convinced that they are impotent, that their complaint can be read in their faces, that they can never marry, and that their future is ruined. Sometimes hypochondriasis takes the form of a morbid dread of syphilis in one who has exposed himself to the risk, but has never had the slightest indication of infection; every sensation about the genitals, or ache or pain in other parts of the body, is put down to the dread disease, and no assurances to the contrary have any effect. In other cases cranial sensations are the prominent feature, and the sufferer fears tumours of the brain, or madness.

Hypochondriasis may last for years, with intervals of improvement. Occasionally, it results in definite insanity of melancholic type; but in its ordinary forms it must be distinguished, if possible, from melancholia. The true hypochondriac does not have delusions, and is not suicidal. The diagnosis of hypochondriasis rests upon the manner of the patient and the nature of his complaints combined with the absence of all serious disease, which should, of course, be carefully searched for. Lesions may sometimes be present which will not, however, account for the intensity or abundance of the symptoms.

Treatment.—This is in great part moral; the ailments must not be entirely ignored, nor must their importance be confirmed by too numerous prescriptions; the patient must be induced to turn his mind to other matters, to associate with those who may distract his attention from dwelling upon his own health, or to seek variety in travel or other healthy recreation. Any defect in health, such as anæmia, constipation, indigestion, or the gouty state, should receive appropriate treatment.

NEURALGIA.

This term is used for a special kind of pain, felt in the course of a particular nerve and its branches, and not referred solely to the peripheral extremities of the nerve. Similar pains are undoubtedly sometimes caused by direct lesions of the trunk of the nerve, such as the pressure of a tumour, or the existence of neuritis; but it is desirable, as far as possible, to exclude these from the category of neuralgia, and to limit the term to the cases of purely functional disturbance.

Ætiology.—Neuralgia is a disease of early adult and middle age, being most common between twenty and sixty. It is more frequent in women than in men, but not very largely so. First among the predisposing causes is the neuropathic disposition: patients are nervous, excitable, or have a family history of insanity, hysteria, epilepsy, or other nerve-complaints. Rheumatism and gout are also frequent antecedents of neuralgia. The exciting agents are depressed health from any cause, such as deficiency of food, over-lactation, and anæmia; physical fatigue, and depressing emotions; exposure to cold, especially the direct incidence of a draught of cold air upon the nerve concerned; peripheral irritation of the branches of the nerve itself, or of a neighbouring nerve, as seen in neuralgia of the fifth cranial nerve from the irritation of a carious tooth. Certain toxic conditions are influential in the causation of neuralgia—namely, alcoholism, lead-poisoning, malaria, and diabetes.

Symptoms.—The pain of neuralgia is deep-seated, and corresponds pretty closely to the position of a nerve-trunk, spreading along its course, or radiating with its branches. It is accordingly often one-sided, but it may be bilateral, and even symmetrical. In character it is variable—shooting, stabbing, boring, burning, gnawing, or throbbing. In its duration it varies considerably; it either lasts only a short time, for minutes or hours, and then subsides entirely, to return after an interval of weeks or months; or it is continuous over long periods in a moderate degree, with exacerbations of great severity from time to time. After it has continued in one of these forms for some little time, the *tender points* of Valleix may usually be recognised; these are spots on the surface of the skin, which are tender to firm pressure. They lie in the course of the affected nerve or its branches, and correspond to the point of exit of the nerve from a bone, or where it perforates the fascia, or where it passes over a hard surface, or where the nerve divides into two branches, or where two nerves anastomose. Another tender spot is less frequently found—the *apophysial point* of Trousseau; this is over the vertebral spine which corresponds to the nerve affected with neuralgia.

There is often some tenderness of the skin over the painful part, which may last for a time after the subsidence of the pain. Occasionally some muscular spasm takes place as a reflex effect in the region of the affected nerve, and vasomotor disturbances may be present, such as pallor at the beginning of the attack, followed by flushing, sweating, lachrymation (in trifacial neuralgia), and œdema. The hair may change colour, or fall off, or, more rarely, it grows in excess.

Some special forms of neuralgia may be more fully described.

Neuralgia of the Fifth Nerve (*Trifacial or Trigeminal Neuralgia; Prosopalgia; Tic Douloureux*).—This may affect either one branch of the fifth, or two of its branches; or the whole of the sensory division of the nerve.

When the *first* division is affected, the pain is over the forehead, the anterior half of the scalp, the eyelid, eye, and side of the nose (*supra-orbital neuralgia*, or *brow ague*). Tender points are found at the supra-orbital notch, at the outer side of the eyelid, or at the side of the nose, and sometimes there is an *ocular* point within the eyeball.

If the *second* division is attacked the pain extends over the cheek between the orbit and the mouth, and to the ala of the nose. Tender points are found at the infra-orbital notch, the side of the nose, on the prominence of the malar bone, and along the line of the gums below it.

If the *third* division is affected, the pain spreads over the parietal eminence, the temple, the ear, the lower jaw, and the tongue. The chief tender points are over the dental foramen, and over the auriculo-temporal branch of the back part of the temple, or just above the zygoma in front of the ear.

The pain is often exceedingly intense, lasting but for a few minutes, and recurring at irregular intervals. It may radiate from one branch of the nerve to another, or to some other nerve. In the most severe cases, the facial muscles are seized with spasm during the height of the pain, and the vasomotor symptoms, such as flushing, local sweating, lachrymation, discharge of nasal mucus, and salivation, are well marked. Increased sensitiveness to sounds, and flashes of light in the eyes, may also occur. The attacks are often brought on by cold, and especially by mastication, so that in some cases feeding becomes most difficult.

A *cervico-occipital* neuralgia occurs with pain in the region of the upper four cervical nerves, and over the back part of the [illegible] Tender points are found where the great occipital nerve [illegible] superficial, in the posterior triangle over the brachial [illegible] and over the parietal eminence; the last is common to [illegible] and trigeminal neuralgia. Occipital neuralgia may be [illegible] by disease of the teeth. It is often bilateral, and the pain is more often continuous with exacerbations, than truly intermitting.

Cervico-brachial and *brachial* neuralgia occur with pain extending over the area of distribution of the brachial plexus, and tender points most commonly in the axilla, at the posterior border of the deltoid, behind the elbow (superior ulnar), and especially one in front of the wrist (inferior ulnar).

Intercostal neuralgia is generally more or less continuous with acute exacerbations; the pain takes the course of an intercostal space, and tender points are found near the spine, in the mid-axillary line, and near the middle line in front.

Lumbo-abdominal neuralgia corresponds to the lower dorsal nerves, and occupies the lower half of the trunk. Tender points are found in positions corresponding to those just mentioned—namely, near the spine, at the middle of the iliac crest, and at the lower end of the rectus muscle. A *scrotal* (or *labial*) point may also be found.

A *crural* neuralgia in the region of the supply of the lumbar plexus is rare. There are some painful affections of the foot, as *painful heel* and Morton's *metatarsal neuralgia*. *Sciatica*, commonly regarded as a neuralgia, is nearly always a neuritis (*see* p. 206).

Some *visceral neuralgias* will be mentioned in the appropriate places.

Diagnosis.—This rests chiefly upon the remittent and intermittent character of the pain, its change from one nerve to another, and the absence of other symptoms indicating any organic lesion of the nerve, or of other parts connected with the nerve. An organic lesion of the nerve, such as neuritis, or compression by new growth, would be suggested by persistent anæsthesia, atrophy of muscles, or lasting trophic changes. A very long duration would, on the whole, be in favour of neuralgia and against organic change. In all cases the evidences of diseases competent to produce pain in the region of the nerve affected should be carefully sought for. The lesions likely to produce this effect vary of course in the different parts of the body. Carious teeth may cause genuine toothache, or without local pain may cause real neuralgia in every division of the fifth nerve, or in the occipital region. Disease of the bones and periosteum, and deep-seated tumours, may involve the main branches of nerves. The cervical and brachial nerves are involved in caries and new growths of the cervical spine. The brachial nerves may be wounded, but are often the seat of neuritis. Intercostal pains may be due to disease of the ribs, caries and cancer of the vertebræ, spinal meningitis, tumours, and aneurysm. A bilateral pain is common in locomotor ataxy. Disease of the lumbar vertebræ, renal colic, and renal disease are responsible for many pains in the area of the lumbar plexus, and sciatic pains are produced by disease of the sacro-iliac joint, and of the hip joint, by psoas abscess, pelvic tumours, and tumours of the femur. Locomotor ataxy here also produces its shooting and stabbing neuralgic pains, which are generally bilateral, and accompanied by other indications, such as loss of knee-jerks, loss of pupil light-reflex, and impaired sensation in the feet.

The **Pathology** of neuralgia is as yet obscure, but there is much in favour of the view that it is a functional disturbance of the sensory nerve-cells.

Treatment.—This includes the removal of the cause if it can be ascertained, the improvement of any general ill-health which may excite or predispose to it, and the use of such drugs or methods of treatment as may modify the morbid condition of the nerve or centre. As we are not using the term neuralgia for the pains of neuritis or nerve-compression, the *removal of the cause* can only apply to those sources of irritation, such as carious teeth, by which a reflex pain is produced. Though this may be effected, it does not follow that the neuralgia will at once cease; the altered condition of the nerve-centre still counts for something, and the pain may be only lessened until other remedies are called in to assist. This fact should make one very cautious in recommending the removal of teeth, unless it is certain that the neuralgia is wholly or chiefly dependent upon the offending member. Many patients have submitted to the loss of all the teeth on one side of the jaw without any material benefit. The *general treatment* of the patient consists mainly in the administration of good food, and of tonics, such as cod-liver oil, quinine, iron, nux vomica, and strychnia. The late Dr. Anstie strongly recommended cod-liver oil as a fat, and if that could not be taken, he prescribed butter, cream, Devonshire cream, or olive oil. Among the remedies which are especially *directed against the local disease* are the following: Arsenic, in full doses (5, 7, or 10 minims of the liquor), or small doses gradually increased, especially in malarial cases; ammonium chloride, in doses of 15, 20, or 25 grains, three times daily; potassium bromide, in full doses; butyl-chloral hydrate, in 5 or 10 grain doses, especially in neuralgia of the fifth nerve; tincture of aconite in 2 to 5 minim doses; tincture of gelsemium, in 15-minim doses; and exalgin in 1 to 3 grain doses. Cannabis indica, ether, valerian, turpentine, and nitro-glycerin are sometimes useful; and a small dose of brandy or wine will often give much relief, but it is obvious that its use should be indulged in with much caution, since drinking habits may easily result from it under these circumstances. The same may be said of opium or morphia, of which the latter may be given in subcutaneous injection, but the dose should be small ($\frac{1}{12}$ to $\frac{1}{6}$ grain), especially at [illegible] the possibility of the morphia habit being induced should [illegible] sight of. Half a grain to a grain of cocain may be injected locally. Some relief may also be got by local external treatment, such as the application of various anodyne liniments (aconite, belladonna, opium, or chloroform), of menthol, and of the ointment of veratria or of aconitia. Counter-irritation may be applied by mustard or blisters; and the actual cautery has been used, especially in spinal neuralgia.

Electricity is sometimes a benefit. A strong current, either faradic or galvanic, acting as a sort of counter-irritant, does good in some recent cases of hysterical neuralgia; more often a weak current seems advisable to act as a sedative to the nerves. For this a strength of from 2 to 5 milliampères should be used with the anode on the seat of pain, and the kathode in some other part of the body, the direction of the flow being a matter of no importance. A very weak faradic current may serve the same purpose.

Finally, in extreme cases, when all other methods have failed, neurotomy, neurectomy, or nerve-stretching offer themselves as possible means of recovery. Hitherto, however, the results have not been very encouraging; generally there is relief for a time, and then, after some months, a recurrence of the neuralgia as bad as before. Nerve-stretching, as the less severe operation in case of failure, is rather to be preferred.

WRITERS' CRAMP AND ALLIED NEUROSES.

In a number of occupations involving complicated movements for long periods of time, such as writing, piano-playing, violin-playing, using telegraphic instruments, rolling cigars, &c., there is a certain liability for the muscles concerned to contract in a spasmodic and irregular way, so that the movement is badly performed and ultimately cannot be effected at all.

This is most common in those who have a great deal of writing as their daily occupation, such as lawyers' clerks, secretaries, &c. The disease is hence called *writers' cramp* and *scriveners' palsy*; *graphospasm* or *mogigraphia* have been used as technical terms. This affection is naturally more common in men than in women, and occurs mostly between the ages of twenty and forty. Its predisposing causes are a hereditary tendency to nervous affections, and a nervous and irritable habit of body; depressing mental conditions, mental anxiety, or business worry. Sometimes it begins after an injury, or some local disease of the hand or fingers. But the most common antecedent is excessive use of the hand in writing. Gowers points out that in the act of writing the pen may be moved across the paper in four different ways—(1) The little finger is fixed on the paper, and the fingers carrying the pen work upon the little finger as a pivot; (2) the wrist is fixed and acts as the pivot; (3) the pivot is at the centre of the forearm, resting perhaps on the edge of the table, or desk; (4) all the movements take place from the shoulder. In the first method, the movements of the fingers are most complicated and strained; and in the last there may be no finger movements at all. He states that writers' cramp scarcely ever affects those who employ the last two methods of writing.

Symptoms.—The affection generally comes on gradually; it may be felt at first as some degree of aching or strain, which is relieved by ceasing to write. After a time the act of writing is accompanied by a spasmodic tonic contraction of the finger or thumb holding the pen; the finger is pressed firmly on the pen, or it is flexed so as to move up the pen, or it slips off the pen so that the latter is grasped between the fore and middle fingers. The thumb may be similarly affected, or the fingers may be extended or lifted from the paper, or the pen may be driven into the paper, or the hand stops its movements entirely. The attempt to continue writing under these conditions produces a cramped, irregular, angular writing, with thick down-strokes; and after a time the spasm becomes so pronounced as to render the act impossible. This is the *spasmodic* or *spastic* form of Benedikt, which is by far the most common; but sometimes there is tremor of the fingers—*tremulous* form; and a *paralytic* form with fatigue alone has been described, but is quite rare. The spastic form often leads, by the frequent contraction of the muscles, to pains in the hands and wrist, which may after a time become distinctly neuralgic in character; and there is often some tingling or sense of numbness. The spasm may be limited entirely to the act of writing, and other movements, even of a delicate nature, can be performed without difficulty. Sometimes writers' cramp is associated in the same person with spasm on playing the piano or violin, and not infrequently in severe cases some other operation may be at the same time imperfectly performed.

The muscular power is for the most part preserved, or there may be a little weakness of grasp, or slight but definite weakness of certain muscles of the hand (Poore). The electric reactions may be quite normal, or they show a slight increase or diminution of irritability in some old cases.

The course of the disease is variable. In slight cases treated at once by perfect rest from writing, the patient may recover completely; but if he has persevered, forcing himself to write by steadying his hand with the other, or by mechanical contrivances, and has ignored all treatment, the disease is often quite obstinate, and may never be thoroughly cured.

The Diagnosis is not generally difficult: writers' cramp at least is not likely to be mistaken for anything else, but it must be remembered that some nervous diseases, such as chorea, hemiplegia, and other paralyses involving power in the hand, may be first detected in the attempt to write, and may be regarded wrongly as writers' cramp. Nervous people, too, who have obtained some acquaintance with the disease may easily fancy that a little fatigue is the commencement of it.

Pathology.—This affection has been explained as arising in weakness of certain muscles, and the over-action of antagonist muscles; or in weakness of one muscle being supplemented by another muscle, which in its turn gets fatigued and is followed by another, until all are worn out; or as the result of reflex action, stimulated through the sensory nerves. A probable explanation is, that it is due to a defect in the centres associated for the act of writing by a morbid lowering of resistance in the commissural connections between the centres, so that there is a radiation of impulses, and so over-action of muscles not necessarily engaged in the act.

Treatment.—The first essential is complete rest from writing. In mild cases this is sometimes sufficient to effect a cure in one or two months. Gowers then insists that on again beginning to write, the patient should learn to write from the shoulder entirely. In more severe cases a much longer rest is required, and if writing is necessary to the patient, he may learn to write with the left hand or use a type-writer. Occasionally, but by no means always, the newly-educated left hand also becomes affected. Various devices have been invented, or are improvised by the patients themselves, to save the strain on the muscles of the fingers, such as running the pen through a cork, which gives a larger grasp; holding a wooden ball in the hand, upon which the pen is fixed at the required angle. Nussbaum has invented a "bracelet" which carries the pen, and surrounds the fingers, so that they hold it by muscles (abductors) different from those commonly employed in writing. But, as a rule, all these instruments only postpone the time at which complete rest must be taken. A return to the normal condition of nerve- and muscle-action may be sought in the use of general and nervine tonics, such as iron, quinine, arsenic, and strychnia; and in local treatment, such as electricity, gymnastic exercises, and massage. The first is recommended in the form of a continuous current with the anode stationary upon the brachial plexus, or upon the peripheral nerves and muscles concerned, the kathode on the cervical spine. Poore advises the combination of electricity with regulated exercises of the muscles. Passive manipulations and massage of the affected limb have been of late employed in a systematic way by specialists, and, it is stated, with great success.

The treatment of the other occupation-neuroses must be the same in principle as that already described for writers' cramp.

DISEASES OF THE MUSCLES.

MYOSITIS.

INFLAMMATION of muscles is rare as a primary disease. Abscesses occur in the muscles as a part of pyæmia, septicæmia, and glanders; and as a sequela of enteric fever and possibly other infectious processes.

Myositis ossificans is a disease in which the muscles are converted into osseous tissue. It always begins in early childhood, invades first the muscles of the back, and runs a progressive course.

MUSCULAR RHEUMATISM.

(*Myalgia. Rheumatic myositis.*)

This name is given to a painful affection, apparently involving the muscles or fasciæ. The connection with rheumatism is not always obvious, nor is it even certain that the muscles or fasciæ are really involved; but the disorder is often the direct result of damp or cold, or of excessive muscular exertion or strain.

Symptoms.—As a rule, only one muscle or group of muscles is affected at a time; and as certain muscles are particularly prone to it, special names are given to the disease, according to its locality. The symptoms are intense pain on attempted movement involving the muscle, and tenderness on manipulation. The pain comes on rather suddenly, and when it is severe, necessitates the patient assuming a position by which it can be relieved. A slight degree of pyrexia may accompany the illness, which usually lasts a few days, but may in some cases be of longer duration.

The more usual seats of the disease are the following:—(1) Lumbar and lumbo-spinal muscles—*lumbago*. This is common in advanced life, and in men more than in women. The patient walks with difficulty, and in a stooping position; any movement of the lumbar region is painful. (2) Intercostal muscles—*pleuro-*

dynia. Breathing, coughing, and all respiratory movements cause severe pain, so that pleurisy may be suspected. But there is no rub, and the constitutional disturbance is slight or none. (3) Cervical muscles—*rheumatic torticollis.* (4) Muscles of the shoulder—*omalgia.* (5) Muscles of the scalp—*rheumatic cephalalgia.*

Treatment.—Complete rest is desirable, and benefit is derived from local applications, such as hot poultices and fomentations, belladonna and aconite applications. Massage is also of value, and any means by which free perspiration is induced, such as the vapour or Turkish bath. Both the galvanic and faradic currents may also do good. Internally, saline remedies, such as potassium citrate, acetate, or tartrate, may be given in full doses, and potassium iodide, in 5 or 7 grain doses. Salicylic acid in doses of 15 or 20 grains is also useful. The possibility of strained positions unconsciously assumed should be inquired into.

PRIMARY MUSCULAR ATROPHY AND PSEUDO-HYPERTROPHY.

It has already been shown (p. 246) that the muscles may undergo a primary atrophy as well as the atrophy which is secondary to diseases of the spinal cord and of the nerves. The origin of these forms of disease is still very obscure; they show strong hereditary connections, they occur in young people, are more frequent in males, but are often transmitted through the females, and no doubt depend upon a congenital tendency to early degeneration. The muscles selected for first invasion are different from those picked out by spinal disease; sensation is unaffected, fibrillary twitchings are generally absent, and though the response to electrical currents is by no means ready there is no reaction of degeneration.

Several varieties have been recognised; in some the early stage is characterised by enlargement of the muscle, by fatty and fibrous tissue, and pseudo-hypertrophy, to which atrophy eventually succeeds; in others, there is atrophy from the first, and rarely, if ever, the semblance of hypertrophy. Erb groups them all under the term *progressive muscular dystrophy.*

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.

The striking feature of this disease is enlargement of the muscles with loss of power; but the same muscles subsequently become atrophied, and atrophy also occurs in some muscles that have never been enlarged.

Ætiology.—It is much more common in boys than in girls, tends to occur in different members of the same family, and is handed down to succeeding generations, as a rule, through the mother, and not through the father. But since a girl who develops the disease is little likely to become a mother, it is mainly through healthy mothers, the sisters of those affected with the disease, that it is transmitted. Thus, to quote Gowers, "a boy suffered, and his sister, unaffected, had two sons diseased, and a daughter free, of whose children two sons were the subjects of the malady." It commonly shows itself in early childhood, either when the child first begins to walk, or when it has attained the age of five or six. In only a few cases is it delayed to the age of twelve or thirteen, and very rarely to eighteen or twenty.

Symptoms.—It may be first noticeable as a delay in acquiring the art of walking, although the muscles of the limbs are sufficiently large, and apparently well developed; or, the child gets easily tired in walking, or walks unsteadily, and finds a difficulty in getting upstairs. As the child gets older, the condition of the muscles attracts attention. At first one calf, and then the other, is noticed to be large, and subsequently the enlargement may affect the glutei, the lumbar muscles, and those of the trunk, shoulder, and upper arm. The enlarged muscles are unusually hard, firm, resisting, and suggestive of great strength, to which the actual capabilities of the patient by no means correspond. But this condition of apparent hypertrophy is not universal; generally, some muscles are found to be atrophied, and these are mostly muscles in the upper part of the body, or some of those of the arm, especially the latissimus dorsi, the lower part of the pectoralis major, and the teres major; but the crural muscles are also often wasted. On the other hand, the deltoid and the infraspinatus may be enlarged, but the muscles of the face, tongue, neck, forearm, and hands are either not affected at all, or affected very late. The weakness of the muscles produces some defects of motion, which are very characteristic. The patient walks with a waddling gait, the feet widely separated, and the body thrown from side to side with each step; the gait is further modified by the tendency to walk on the toes, which results from the diseased calf-muscles shortening and producing a modified talipes. In standing, the legs are also widely separated for the sake of equilibrium, and the back assumes the position of *lordosis*, or curvature with the concavity backwards. This is because the weakened glutei and extensors of the hip allow the pelvis to drop forwards, and the balance of the body is then only preserved by the shoulders being thrown back so that a line dropped from them falls even behind the sacrum. Another characteristic peculiarity is the way in which the patient rises from the sitting position on the ground. He rolls over on his hands and knees, and, if a chair or bed be near, pulls himself up by its means; but if alone, he lifts the knees from the ground so as to be on his hands and toes; then, swinging himself over towards one side, he places the opposite hand on its corresponding knee, and by its means straightens the leg. The same manœuvre is repeated with the other hand and knee, so that he now stands with legs wide apart and a hand on each knee. With a great effort, then, the back is gradually straightened as the hands are brought higher and higher up the thighs.

The weakness of the spinal muscles is also shown by the inability to pick up objects from the floor, and by the falling forward of the body if the patient is sitting and leans forward too much; further, there is great difficulty in ascending stairs, so that the help of the banisters is sought.

The electrical condition of the affected muscles is not at first much altered. When they have become very weak there is diminution to both faradic and galvanic electricity. There is no degenerative reaction. The knee-jerk is, after a time, diminished, and in advanced cases lost.

Beyond this, the nervous system is normal: sensation is unaffected; the bladder is only affected occasionally towards the end; and the mental functions are mostly unimpaired, though, according to Ross, some degree of mental incapacity and even idiocy may co-exist.

After the first development of the symptoms the disease may remain stationary for two or three years, but the patient gradually gets weaker in the legs, the power of standing is lost, wasting takes place, especially in the muscles of the upper extremities, and the patient is confined to bed. Finally, death from intercurrent illness, such as inflammation of the lungs or an acute infectious disease, takes place about puberty in the majority of cases; but in those that have developed slowly the fatal termination may be correspondingly delayed.

Anatomical Appearances.—These affect the muscles only. The spinal cord and nerves have hitherto been found practically normal. The muscles affected with pseudo-hypertrophy look to the naked eye like masses of fat, and under the microscope are seen to consist largely of adipose and fibrous tissue, with muscular fibres thinly scattered throughout. The change begins by an increase of the connective tissue between the muscular bundles. Fat then develops in this connective tissue, and the muscular fibres are pressed upon and atrophy. In the process of atrophy the transverse striæ may become fainter, or be replaced by fatty granules, or by longitudinal striation. But some fibres remain normal in size and striation, though separated from one another by broad tracts of fat and connective tissue. Other fibres are diminished in size or irregular in diameter; and many have obviously disappeared. In some muscles the development of fat is not so marked, and the colour is better preserved, but there is a large excess of fibrous tissue.

Diagnosis.—This is not generally difficult. The prominence of the calf muscles, associated with weakness, difficulty in walking and in rising from the ground, and lordosis, are commonly sufficient to distinguish it. Gowers lays more stress upon the enlargement of the infra-spinatus together with wasting of the latissimus dorsi and the lower part of the pectoralis major. The *spastic paraplegia* of children may resemble it, as the calf muscles may be contracted, firm, and of good size; but this contraction is active and can be overcome, the knee-jerk is excessive, the muscles show a tendency to spasm, and the children do not rise from the ground in the way peculiar to pseudo-hypertrophic paralysis. In *spinal atrophies* the distribution of the order of wasting is somewhat different, and there is no enlargement at any time.

The **Prognosis** is very unfavourable, and **Treatment** can at best retard its progress a few months or years. Drugs probably have no influence, and electricity little, if any. Gowers thinks that carefully planned gymnastic exercises have done good, and further recommends rubbing, massage, and passive movements. When the gastrocnemii are so shortened as to prevent the patient standing, the tendons should be divided.

Idiopathic Muscular Atrophy.

Under this name are grouped some allied forms of disease in which muscular atrophy is the important characteristic, or exists alone. Like pseudo-hypertrophic muscular paralysis, idiopathic muscular atrophy occurs in several members of the same family, but it shows no preference for boys over girls. Weakness and wasting come on simultaneously, and are first noticed in some cases in the face, in others, in the upper extremities, and in others, again, in the lower extremities. Different types have been recognised by writers, but they are all probably allied to each other, as well as to pseudo-hypertrophic paralysis. It will be sufficient here to mention two of these.

Erb's juvenile form.—This commences usually about the age of puberty, and affects the muscles of the upper extremities first. The biceps, triceps, and supinator longus are atrophied, and the latissimus dorsi, the lower part of the pectoralis major, the serratus, trapezius, and rhomboid also suffer, but the deltoid, supra-spinatus, infra-spinatus, and subscapularis generally escape. The muscles of the forearms are but little affected, and the small muscles of the hand rarely. The spinal muscles vary in different cases; the intercostals, the diaphragm, and the abdominal muscles are sometimes affected. Of the leg-muscles, the glutei, the flexors of the hip, and the extensors of the knee are most often diseased, and the muscles below the knee often escape; but the peronei are atrophied occasionally. A temporary enlargement may occur in some muscles, especially the deltoid, infra-spinatus, and the muscles of the calves. If the disease begins in the legs, it is likely to be at an earlier age than when it begins in the shoulders and arms; and in either case the facial muscles rarely suffer.

Infantile form or facio-scapulo-humeral type.—This begins at a very early age, and the facial muscles are first affected. The zygomatici fail early, the naso-labial fold is lost, and in smiling the angles of the mouth are drawn outwards instead of upwards and outwards. The orbicularis oris is weak, so that the lower lip drops, the mouth "pouts," and labials are badly pronounced. The orbicularis palpebrarum is occasionally affected, and the eyes remain unclosed; or the frontales are paralysed, and the forehead cannot be wrinkled. The tongue, the larynx, the pharynx, the muscles of mastication, and those of the eyeballs are never involved. Only later are the muscles of the shoulder, arms, hand, and perhaps of the legs, implicated.

In all these forms, fibrillary twitchings are, as a rule, absent. Irritability is diminished equally to both electric currents, and there is no reaction of degeneration.

Deformities may occur similar to those of pseudo-hypertrophic paralysis, such as lordosis and talipes. The course of the disease is variable. It may remain limited to the muscles first affected, or at intervals of years it may spread to others. Its duration may be from ten to fifty years; and death takes place from intercurrent affections, such as phthisis. Hitherto, only a few *post-mortem* examinations have been made. The muscles in one case were found to be small and pale, with intense granular and fatty degeneration of the muscular fibres; in another there was simple narrowing of the fibres. The spinal cord and nerves were normal.

Treatment.—Electricity, massage, regulated voluntary exercise, and attention to the general health are recommended in order to retard as much as possible the progress of the disease.

THOMSEN'S DISEASE.

This very rare disease, of which hitherto only a few cases have been recorded in England, appears to be a form of muscular dystrophy, and may properly be here considered.

It consists of a peculiar rigidity of the muscles, which comes on whenever they are called into contraction by voluntary impulses after a period of rest. Thus, if the patient wishes to walk and tries to rise from his seat, his muscles become rigid, and he is unable to move; the rigidity lasts a few seconds, and then relaxes so that he can at length get up. His first few steps are attended with the same difficulty, but soon the contractions become more natural, and shortly the trouble ceases altogether, so that he walks with complete freedom and ease. If, however, he should stop for a minute, the muscles become rigid on beginning to walk again. Quick movements are thus impossible immediately after rest, and sometimes accidents occur, as, for instance, when the patient is descending from a train, and having placed one foot on the ground, he is unable to bring the other out quickly after it, and falls in consequence. The muscles of the lower extremity are most affected, but those of the arms, and even those of the face, are sometimes involved to a less degree. The tendency to rigidity seems to be increased by cold and by mental anxiety. The following changes are noticed in the muscles: they are hypertrophied, but their power of contraction is rather diminished; contraction to direct mechanical stimulation (idio-muscular contraction) is said to be in some cases exaggerated; the closure contraction to strong galvanic currents is much prolonged beyond the time of application, and with continued application of the current, a series of contractions has been seen to pass in a wave-like manner from the kathode to the anode. The reactions to faradism are often normal, but may be exaggerated in force and duration. The disease is often of very long duration, but may subside.

Ætiology.—It happens often early in life, may affect more than one member of a family, and is commonly regarded as being congenital (*myotonia congenita*).

Pathology.—In a case reported by Déjerine and Sottas, the patient died of nephritis. The muscles were large, protruding, tending to be more globular in shape, of a duller red colour, and less elastic than normally. The number and size of the muscle-nuclei were found to be increased, the fibres varied much in size, some being very large indeed, as if swollen. Sometimes the sarcous elements were separated and the sheath was filled with granules; or the muscle substance was degenerated and vacuolated. The connective tissue was not hypertrophied or fatty as in pseudo-hypertrophic paralysis. These results agree closely with what had been seen in portions of muscle excised during life. The muscle-nerves, nerve-trunks, spinal cord, and bulb were healthy.

Treatment.—Nothing has been found of use. Thomsen, who suffered from it himself, advised a life of continued activity.

DISEASES OF THE ORGANS OF RESPIRATION.

PHYSICAL EXAMINATION OF THE CHEST.

Since the lungs are contained almost entirely within the bony thorax, or chest, the diseases of these organs are likely to reveal themselves by modifications in the shape, in the movements, and in the acoustic phenomena yielded by the chest. A consideration of these various physical signs or indications of lung diseases must precede their systematic description.

For purposes of accurate description it is necessary to divide the surface of the chest into regions. Front and back, right and left, suggest themselves at once, but these are too extensive. Some writers have drawn horizontal and vertical lines, and given different names to the quadrangular areas thus indicated, but in practice it is not easy to remember the exact positions of the lines. The same purpose is sufficiently served by a reference to certain points or surfaces in front or behind, the regions being named according to their situation in relation to these points. Thus we have the sternum, the clavicles, and the nipples in front, the scapulæ behind, and the axillæ at the side, and we may recognise accordingly the following regions: From above downwards on each side in front, supra-clavicular, clavicular, infra-clavicular, mammary and infra-mammary; in the middle line supra-sternal, upper sternal, mid-sternal, and lower sternal; at the side, axillary and infra-axillary; behind, supra-spinous, infra-spinous, infra-scapular, and inter-scapular. These occur on both sides of the body, except the sternal regions, which are in the middle line. Such regions, it is true, are not perfectly defined, but sufficiently so for many observations. Where perfect accuracy is called for it can only be obtained by stating on which rib or intercostal space the point under investigation is situated, and how far from some fixed line or point, like the middle line, the edge of the sternum, the angle of the rib, or the nipple.

The modes of examination of the chest are inspection, palpation, mensuration, percussion, and auscultation.

INSPECTION.

By simply looking at the chest in front, behind, and from above, any alteration in its shape and movements can be detected. The chief points to be noticed in a healthy chest are as follows:—It has a somewhat flat oval form—that is, the antero-posterior diameter is much less than the transverse; its greater breadth is at the lower part; the clavicles are only slightly prominent, with but little depression above, and scarcely any below them; the position of the nipple is on the fourth rib, or on its upper or lower border; the angle (*epigastric angle*), which has its apex at the ensiform cartilage, and is bounded on each side by the seventh and eighth costal cartilages, is from 95 to 105 degrees; the scapula is closely adapted to the posterior part of the thorax; and the spine is straight. In inspiration, the chest should expand from 1½ to 2½ inches in circumference, the two sides should move symmetrically, the epigastric angle should be widened, the sternum thrown forwards, the lower ribs lifted, and there should be only very slight recession of the lowest intercostal spaces on deep breathing.

By inspection in disease we may see that one side is larger or more contracted than the other, that the movement is deficient on one or other side, or that the intercostal spaces are unduly sucked in; and we may also see at once the position of the heart, which may be disturbed by disease of the lung.

The deformities of rickets and of angular and lateral curvatures, which are not due to diseases of the lungs, but may seriously impair the action of these organs, should be specially noted.

By inspection also, apart from changes in the shape and symmetry of the thorax, we may note the character of the respiratory movements. The normal frequency of respiration in adults is from 15 to 18 in the minute; in children it is much more rapid, and varies with the age. In different forms of disease, both pulmonary and of other origin, the movements may be slower or more rapid, shallower or deeper, feebler or stronger than is normal, and they may be irregular. The term *dyspnœa*, meaning difficult or bad breathing, is more generally used to indicate that the breathing is unduly rapid—shortness of breath—or that it is performed with effort. The necessity for such effort may lie in some obstruction to the passage of air, or in defective muscular power. The difficulty may be most marked during inspiration (*inspiratory dyspnœa*), or during expiration (*expiratory dyspnœa*). If the patient is obliged to sit up in order to breathe, as is the case in many pulmonary and cardiac diseases, the condition is called *orthopnœa*.

It is important also to note whether the breathing is effected more by the upper part of the chest, which is normal to women, or by the lower part and the diaphragm, which is more characteristic of men. The inspection must be extended to the abdominal walls, which reflect, so to say, the action of the diaphragm, advancing when it contracts and receding as it relaxes. A disproportionate use of one part of the chest suggests disease in another part. The inspection should be made both during quiet breathing and when the patient makes a forced inspiration.

A peculiar form of breathing is known as *Cheyne-Stokes' respiration*. It is characterised by alternating periods of very rapid and very slow movements. As the movements get slower there is at length a pause of several seconds, then the movements begin again, very feeble at first, then quicker and stronger, until they may occur at the rate of 50 or 60 per minute; then they rapidly slow again, and the cycle is completed by the long pause. The whole duration of a cycle may be from 20 to 60 seconds, and the number of respirations in a cycle varies from five to sixty. In some cases the changes in the breathing are accompanied by other curious phenomena. For instance, in the stage of rapid respiration, the patient may become excited, groan, struggle, or even try to get out of bed; as the breathing slows he becomes quiet, drowsy, and apathetic. More often the pupils dilate in the rapid stage, and contract again in the slow period. Cheyne-Stokes' respiration has not been fully explained, but it is attributed to exhaustion of the respiratory centre. It occurs under many conditions, but most frequently in diseases of the brain and heart, in aneurysm, and in uræmia. It often precedes death only by a few hours or days; but it has been known to persist for months and even years; and it may subside entirely.

PALPATION.

By this is meant the act of laying the hand upon the surface of the chest, either to test its movements or to study the vibrations of its walls produced by the voice or other cause. For the former purpose a hand is laid at the same time on each side below the clavicle, or in the infra-scapular or infra-axillary region, when the absolute and relative amounts of movement can be gauged with some accuracy. For the latter purpose the hand is placed flat upon the chest in different parts successively, and the patient speaks in a loud, clear voice. In health the chest-wall is thrown into vibrations which are plainly perceptible to the hand laid upon it (*tactile vocal fremitus*, or *tactile vibration*). For this it is necessary that there shall be a normal vibration of the vocal cords, and normal conductivity of the lungs with patent bronchial tubes and spongy lung-tissue. The amount of vibration differs in healthy people; it is greatest in adult males with deep sonorous voices; it is least, or it may be absent, in females and children. In disease it is diminished or abolished by anything which obstructs the bronchial tubes or compresses the lung, so as to convert its spongy tissue into solid—*e.g.*, liquid in the pleural cavity. It is increased under some conditions of consolidation of the lung-tissue with patency of the bronchial tubes, especially pneumonia.

By palpation also can be recognised the vibrations of pleural friction, of bronchial narrowing (rhonchi), and of some sounds produced in cavities. The corresponding sounds are described under Auscultation.

MENSURATION.

The chest may be measured in various ways. The ordinary tape measure gives the circumference, and if measurements be taken during expiration and full inspiration, the difference will give a rough idea of the expansion of the chest, or vital capacity. The tape should be applied opposite the nipples. By *callipers* the transverse and antero-posterior diameters can be estimated. The *cyrtometer* consists of two long pieces of soft metal, joined loosely together by one end of each. The point of junction is applied to the spine, and the metal rod on either side is wrapped round the side of the chest at any desired level, so as to take a mould of its shape or curve. The instrument is then carefully removed, without disturbing the moulded curve, and, if it is laid out on a large sheet of paper in the position it occupied while applied to the chest, a pencil can be traced round it, and a permanent record of the shape of the chest is thus obtained. The *perigraph* is another recording instrument invented by Graham Brown.

The movements of the chest-wall can be registered on a dial or paper by the *stethograph* of Riegel and the *thoracometer* of Sibson. The *spirometer* of Hutchinson records, in cubic inches, the air which is breathed out of the chest; the fullest possible expiration after a deep inspiration gives the *vital capacity* (complemental, tidal, and supplemental air together), and this has been found to have a close relation to the height of the individual. It is on an average 174 cubic inches for a person five feet high, and rises 8 cubic inches for every inch of height. Waldenburg's *pneumatometer* measures the force of inspiration and expiration by means of a mercurial manometer. The inspiratory force raises from 70 to 100 mm. of mercury, and the expiratory force from 90 to 130 mm.

PERCUSSION.

In percussion the chest is struck with the fingers or with an instrument called a hammer or *plessor*, so as to elicit a sound. In *immediate* percussion the chest is struck directly with the hammer or with the tips of the fingers, generally the fore, middle, and ring fingers, of the right hand. In *mediate* percussion a finger of the left hand or a small piece of ivory or other material, constituting a *pleximeter*, is laid upon the chest, and this is struck with the finger or hammer. An advantage in using the finger as the pleximeter in mediate percussion is that the vibrations of the chest can be felt at the same time that the sound is heard.

Percussion over the healthy lung elicits a sound, which varies in different parts of the chest, but which has the general characters of what is known as *pulmonary resonance.* It is a rather full and low-pitched note. It can be obtained on the right side from just above the clavicle to the upper portion of the sixth rib; over the whole of the sternum; on the left side from above the clavicle to the upper border of the fourth rib internal to the nipple, and down to the sixth rib, where it passes into the resonance of the stomach. In the right lateral region it extends from the axilla to a horizontal line cutting the eighth rib in the mid-axillary line; on the left side the axillary resonance is limited below by the upper border of the ninth rib. Posteriorly, the chest is resonant from the apices to the lower border of the eleventh rib on the left side, and to its upper border on the right side. The resonance extends a finger's breadth lower than these limits on deep inspiration. The fulness and loudness of the note are most marked in the second intercostal spaces in front, and over the infra-scapular regions behind. Over the clavicle and sternum it is less full, and of higher pitch; and over the supra-spinous fossæ the note is often deficient, especially in very muscular or fat people. At its lower margin the pulmonary resonance is less marked, and approximates to the dulness of the parts below; it is called *transitional* dulness. This extends on the right side in a line above the liver, occupying the fifth space in front, and the seventh in the axillary line; on the left side the line runs from the sternum along the third space, turns vertically down within the nipple to the heart's apex, and may be again noticed in the eighth space just above the spleen. Round the heart this corresponds with the *deep cardiac dulness.*

The healthy percussion note is due to vibration of the chest-walls, and of the columns of air in the lung beneath the point struck. The percussion note varies in healthy persons according to the thickness of the parietes (fat or muscle), and is modified in disease by alterations of the tissue of the lung.

The *intensity* of the note is diminished by solidification of the lung substance. There is then said to be impairment of resonance, or *dulness*. Much overlying fat or muscle will also diminish the sound on percussion. The intensity is increased by the lung-tissue becoming more open in structure, and less finely spongy. This happens in emphysema, and the note is called *hyper resonant.*

The *pitch* of the note is raised by an increase of tension in the chest-wall, by an increase of tension in the lung-tissue, and by a less length of the underlying air columns. It is of course lowered by the converse conditions. It frequently happens that diminution of intensity coincides with elevation of pitch, when the solidification of a portion of the lung shortens the columns of vibratile air under the part percussed.

A *tympanitic* or drum-like note is often observed very similar to that which may be obtained by percussing the distended stomach. This is a purer note than the normal percussion-note, and is due to vibrations taking place in a single large unbroken space. It occurs over very large cavities in phthisis, and in pneumothorax, where one pleural cavity is distended with air. A somewhat similar note, but higher-pitched, is heard over the upper part of the lung in cases of pleuritic effusion occupying the lower half or two-thirds of the chest. It is known as *Skodaic resonance*, and is probably due to partial compression, relaxing the tissue of the lung, and thus giving it, so far as vibrations are concerned, some of the characters of a large continuous cavity.

The *resistance* or want of vibration of parts which are not resonant can also be appreciated by the finger used as a pleximeter.

AUSCULTATION.

This is the study of the viscera or other parts of the body, by listening to the sounds that are produced within them. It may be *immediate*, when the ear itself is applied to the body, or *mediate*, when an instrument is interposed between the body of the patient and the ear of the listener. The instrument is known as a *stethoscope*, and is used in a great variety of forms. Those in most common use are (1) the straight wooden or metal stethoscope, about eight inches long, with an expansion at each end for the chest and ear respectively; and (2) the binaural stethoscope. The latter has the advantage of flexibility, and can be used in all positions of the patient.

By auscultation of the lungs we study the character of the breath-sounds, the transmission of the voice through the chest, and the transmission of the cough.

Auscultation of the Breath-Sounds.—If the healthy lung is auscultated, one hears everywhere, with each respiration, a sound which is known as the *normal breath-sound*, or *vesicular murmur*. It may be imitated by blowing softly, with the lips placed in the position to pronounce the German "w" or English soft "v." As its name implies, it has been regarded as due to vibrations produced in the air-vesicles, as the air passes from the minute bronchial tubes into the wider alveolar spaces beyond; but the vibrations of the air passing between the vocal cords certainly contribute to it. The vesicular murmur is heard during inspiration, but the expiratory act is either quite silent, or is accompanied by a similar sound, much softer, and much shorter in duration. In certain parts of the chest the vesicular murmur gives place to a sound having the characters which will be presently described as those of *bronchial breathing*. These parts are the upper end of the sternum, the first costal cartilages at their junction with the sternum, and a diamond-shaped space at the back in the middle line, including the seventh cervical and first dorsal spine. Elsewhere the vesicular murmur is always present, as long as the lung is healthy and the air-passages are pervious. In children the vesicular murmur is louder than in adults.

Diminished vesicular murmur, deficient entry of air, or absence of breath-sound, occurs if the air-vesicles are obliterated by pressure, or displaced from the surface of the chest, or if the bronchus communicating with them is obstructed or obliterated.

Increased vesicular murmur happens (1) over both lungs from hurried respiration; (2) over one lung or part of a lung, when another part of the lung is not properly in use. It is then called *compensatory* or *supplementary* breathing. It is louder and harsher than the normal breath-sound, and the expiratory murmur is almost or entirely suppressed.

Interrupted breathing.—In this the inspiratory murmur is jerky or wavy, from irregular expansion of the lung, of which the cause may be mechanical obstruction to the entry of air, irregular muscular action from nervousness, or the cardiac impulse. The term *cog-wheel respiration* is sometimes used.

Bronchial breathing or *tubular breathing*.—This modification of the breath-sounds has the following features:—The inspiratory and expiratory sounds are of equal length; they are distinctly separate from one another; they are of higher pitch, and hollower than the vesicular murmur. The sound may be imitated by placing the mouth and tongue in the position to pronounce the German "ch," and then blowing in and out; but there may be very considerable variety in both the pitch and hollowness of sounds that may still all be called bronchial. Such double hollow sounds are heard normally over the larynx, over the trachea, and over the origins of the larger bronchi, at the top of the chest, as already stated; but if they are heard in other parts of the chest they are due to modifications of the lung tissue, mostly a conversion of the spongy lung tissue into solid lung tissue, either by filling up of the air-cells (pneumonia, phthisis), or by compression from without (pleuritic effusion). The necessary condition seems to be patency of the bronchial tubes with consolidation of the lung between the bronchus and the surface. Very different views are still held as to the cause of the sound. It is obvious that the vesicular element of the breath-sound is abolished. One of the earliest views was that the glottic sound was conducted to the surface by the solid lung; another view is that the glottic vibrations are more perfectly conducted along the tubes, dissipation being prevented by the solid lung (H. Mackenzie); another is that the glottic vibrations are resonated in the tube of the affected part; another, that the otherwise quiescent column of air in the bronchial tube of the consolidated part is set in vibration by the air currents moving across its mouth in the larger tube with which it communicates (Bullar). Bronchial breathing may also be produced in small cavities, and in dilated bronchial tubes. Its pitch and hollowness are determined by the length or size of the tube or cavity in which resonance takes place; the higher-pitched, "whiffing," varieties occurring in the narrower or shorter tubes, the lower-pitched in the larger tubes.

Cavernous breathing.—By this term is meant a very hollow breath-sound, in other respects like bronchial breathing. Such hollow sounds often take place in connection with rather large cavities, but there is no broad line of difference between cavernous breathing and hollow bronchial breathing; and breath-sounds which deserve the name of cavernous are often heard over solid lung.

Amphoric breathing.—This is a still more hollow, double sound with a peculiar metallic or ringing character, such as may be produced by blowing softly into the mouth of a narrow-necked glass jar or vase. In its strictest sense it is rarely heard, and then only in very large cavities, or in pneumothorax.

It is important to note that loudness is not a necessary feature of either bronchial, cavernous, or amphoric breathing; the latter especially is often quite soft.

Adventitious Sounds.—The word adventitious expresses the fact that these sounds may generally be heard in addition to, and at the same time as, the ordinary breath-sounds or the breath-sounds modified as above. The adventitious sounds are *rhonchi*, *stridor*, *râles*, and *friction-sounds*.

Rhonchi are more or less musical sounds, due to obstruction of the bronchial tubes, by accumulation of mucus, thickening of the mucous membrane, or spasmodic contraction of their muscular fibres. The sounds vary very much according to the size of the bronchial tube and the extent of the narrowing, and are likened to various familiar sounds, such as cooing, groaning, snoring, grunting, or whistling. The lower pitched, snoring sounds are called *sonorous* rhonchi, and are produced in the larger tubes; the higher pitched, whistling sounds are called *sibilant* rhonchi, and are produced in the smaller tubes. They may be heard with expiration or inspiration, and are constantly changing in position and loudness. Loud sonorous rhonchi are often audible to those standing near the patient, and constitute "wheezing."

Stridor is a loud, harshly musical sound, which is produced by constriction of the glottis, trachea, or one main bronchus. It is less changeable than rhonchus, is audible over the greater part of the chest, and can sometimes be heard by those near the patient without the aid of the stethoscope.

Râles are various forms of crackling or rattling sounds, which are produced in the medium-sized and smaller bronchial tubes, or in pulmonary cavities, by the air forcing its way into fluid secretions accumulated there, and thus causing bubbles to form and burst with a slight noise. They are sometimes distinguished as *moist* sounds, from rhonchi or *dry* sounds; but this is undesirable, if rhonchi may themselves be due to the presence of mucus. The râles differ according to the size of the bubbles, and are called *small*, *medium*, and *large*. Râles are also divided into *bubbling* and *crackling;* the latter have a sharp, clear, ringing, explosive character, which is probably due to their occurrence in the midst of consolidated lung, and to consequent special conditions of resonance; the former, or bubbling râles, are dull, not ringing or explosive, and occur mostly in tubes surrounded by normal spongy tissue. Crackling râles are sometimes called *consonating* from their supposed acoustic origin; and bubbling râles, *non-consonating* in contrast.

Thus we have râles which are

small, medium-sized, or large,	and at the same time	crackling (consonating), or bubbling (non-consonating)

Gurgling is a coarse râle which occurs in large cavities.

Crepitation is a term that has been used indiscriminately for all râles, but is now generally confined to a very fine râle, so fine as to be suggestive of an origin in dry materials (rubbing of hair close to the ear, rustling of silk, or tearing of paper). It is heard in the early stage of pneumonia, in œdema of the lung, and in lung that is forcibly expanded after prolonged collapse. It is probably due to the opening up of minute bronchioles or even air-vesicles that have been adherent by sticky fluid, or from simple disuse. Crepitation and the finer râles are heard only during inspiration; medium-sized and coarser râles may be heard during expiration also.

Metallic tinkling is resonance caused by a râle in a large cavity.

Friction sound, or *pleuritic rub*, is produced by the rubbing together of two pleural surfaces roughened by inflammation. In its most characteristic form it is a rough, grating, interrupted sound, like that which may be heard on forcibly dragging two pieces of leather over one another, or on rubbing the palmar surface of a finger over a wooden surface. It is best heard during inspiration, but may be heard with expiration also.

It must be remembered that any of the above morbid sounds, if not heard during tranquil breathing, may possibly be brought out when the patient takes a deep inspiration.

Auscultation of the Voice.—In most people the voice is transmitted through the chest, and can be heard by the ear or stethoscope placed on any part of it; this is called *vocal resonance*

Diminished or absent vocal resonance.—In children and females with voices of high register, the vocal resonance may be slight or absent. In disease its absence is produced by obstruction of the bronchus, or compression of the lung, involving the bronchus.

Increased vocal resonance.—Bronchophony.—There is naturally a louder vocal resonance at those points where bronchial breathing is normally heard—namely, the sterno-clavicular articulation, and the inter-scapular region (*see* p. 383). In disease, it is caused by consolidation of the lung around patent bronchial tubes, such as occurs in pneumonia, tubercular consolidation, and sometimes compression by liquid. It has been explained as the result of increased conducting power on the part of the solid lung, but the view that it is due to altered conditions of resonance in the tubes of the affected lung is, to my mind, more probable.

Pectoriloquy is the clear transmission of articulate sounds, as distinguished from mere loudness of transmission of the voca vibrations in bronchophony. It may be recognised when the patient speaks loudly, but it is best observed by asking the patient to whisper, when the laryngeal vibrations are absent It is observed over pulmonary cavities, and over consolidated lung.

Ægophony consists of a peculiar *nasal* or *twanging* modification of the voice, heard through the chest. It derives its name from its supposed resemblance to the bleating of a goat. It appear to be due to the suppression of the fundamental tone, and th lower harmonics of the vowel sounds (as pointed out by the lat Dr. Stone), while the higher harmonics are transmitted in as accentuated form, and produce a discordant note. Its mos common cause is undoubtedly the presence of liquid in the pleura and the suppression of the lower tones is attributed to thei inability to pass through—that is, to set up vibrations in—th layer of liquid; but it appears to me, it is better explained by supposing that the bronchial tubes are modified by compression so that whereas normally they *resonate* the lower harmonics, now they can resonate only the higher harmonics; thus the forme are stopped not at the fluid, but at the bronchial tubes. Ægophony is commonly heard at the middle of the back behind, internal to, or below, or over the lower end of the scapula; sometimes over a large area with a vertical extent of several inches, and not necessarily limited to the upper edge of the layer of liquid. It is occasionally heard in front. Sometimes, also, though rarely, it is heard distinctly when no liquid is present, but the condition of the tubes is modified in some other way: thus it may occur in pneumonic consolidation, with a bronchial tube containing fibrin. It is best brought out by asking the patient to utter words containing the vowels *i* and *e*, which depend on the presence of the higher harmonics, such as "three," "ninety-nine."

Auscultation of the Cough.—The patient is directed to cough while the physician auscultates the chest. Increased resonance of the cough occurs under the same conditions as increased resonance of the voice (consolidated lung and cavity); moreover, the cough, and the forced inspiration preceding it, will reveal the existence of râles that are not obvious on ordinary inspiration. In infants the spontaneous cough supplies the information as to vocal resonance, which is given in adults by speaking.

AUSCULTATORY PERCUSSION.

In this process a stethoscope is placed on the chest, and the surface is percussed around it; minute differences of resonance are thus detected. It is not much employed, except in the production of the *bell sound* or *bruit d'airain*, which occurs in pneumothorax. While the physician listens with the stethoscope to one part of the chest, presumed to be the subject of pneumothorax, an assistant lays one coin on the chest, and strikes it with another. The noise is resonated in the hollow cavity, and transmitted as a loud ringing musical note through the stethoscope.

SUCCUSSION.

In cases of hydro- or pyo-pneumothorax, shaking the patient (*Hippocratic Succussion*) will elicit a *splashing* sound, due to mixture of the air and liquid contained in the pleural cavity.

DISEASES OF THE NASAL PASSAGES.

CORYZA.

Coryza, nasal catarrh, or "cold in the head," is so familiar to everyone, that a brief description will suffice. It is a catarrhal inflammation of the mucous membrane of the nose (*acute rhinitis*), which often involves also the conjunctivæ, frontal sinuses, pharynx, and Eustachian tubes, and may spread to the larynx and the bronchial tubes. It is most commonly the result of exposure to cold, either by sitting in a draught, staying out late at night, or by getting wet and failing to change the damp clothes; but it not infrequently runs through a household in a way which strongly suggests that it is primarily infectious in its nature. An attack of sneezing is one of the first symptoms, but this may be preceded by a feeling of indisposition, with chilliness, headache, dryness of the throat, and loss of appetite.

The sneezing is soon followed by the discharge of clear mucus from the nose, which requires the frequent use of the pocket-handkerchief; and there is a feeling of stuffiness in the nose, due to swelling of the mucous membrane. At the same time, the eyes are suffused and water freely, there is pain over the eyebrow from implication of the frontal sinus, the throat is sore, taste and smell are impaired, and there may be deafness from closure of the Eustachian tube. Some febrile reaction is present at the same time. If the catarrh extends to the larynx, the voice is hoarse, and there is constant irritating cough; and its further spread to the lungs will cause the symptoms described below under Bronchitis. After a few days the mucous discharge becomes thicker and more opaque, and may ultimately be quite purulent, continuing thus for a variable period, from two or three days to a week or more. During this time the patient is liable to fresh exacerbations of the inflammation.

Treatment.—In mild forms little requires to be done. Free sweating at night by means of additional blankets, a hot bath, or diaphoretic drugs, often seems to check the disease; if cough be troublesome, a few drops of ipecacuanha wine, with spirits of nitrous ether, or compound tincture of camphor will relieve. Local applications have been much tried of late, either as powder or solution: [illegible], a mixture of bismuth. subnit. ʒvj., morph. [illegible] acaciæ ʒij.; 2 or 3 drachms to be snuffed up [illegible] in the course of a day (Ferrier); or menthol, [illegible] 3 parts, acid. boric., 2 parts. A solution of [illegible] cocaine (2 to 4 per cent.) may be sprayed into the nostrils. Internally, in a pure nasal catarrh, after the first few days, tonics, such as quinine, will do good.

HAY FEVER.

(*Summer Catarrh. Hay Asthma.*)

This is a very severe catarrh which occurs to certain individuals, year after year, in the early part of the summer—that is during June or July, when grasses and other plants are flowering. The symptoms may be chiefly nasal, like those just described, or chiefly bronchial, when the disorder may be called hay asthma. It is undoubtedly, for the most part, due to the diffusion of pollen-grains in the air, and their contact with the nasal, conjunctival, or bronchial mucous membrane, in persons peculiarly susceptible to this form of irritation. Such persons are more often men than women, are generally among the middle and higher classes of society, of neurotic disposition, and have their first attack before the middle period of life. There is sometimes a chronic thickening of the nasal mucous membrane, or chronic hypertrophic rhinitis, which renders the individual constantly liable to catarrhal attacks; and this, or a like morbid condition, is thought by some to be constant and primary. By others the disease is regarded as a part of pulmonary asthma.

Treatment.—Residence in the country during the hay time and exposure to the emanations of grasses must be avoided; if the sufferer goes out, he may wear a veil over the eyes or nose. Locally, M. Mackenzie recommended a spray of a 4 or 6 per cent. solution of cocaine to the eyes; for the nose, the cocaine spray should be followed by the daily introduction along the floor of the nose of a bougie, smeared with vaseline or oil, and left in for ten minutes at first, and for gradually increasing periods up to half-an-hour or longer. He mentions, also favourably, borax and soda washes, the vapour of benzoin, and the insufflation into the nose of morphia ($\frac{1}{16}$ gr.) and bismuth subnitrate (1 gr.). Where there is chronic hypertrophic rhinitis, the application of the galvano-cautery to the swollen mucous membrane, after the preliminary use of a 2 per cent. solution of cocaine, seems to be quickly curative. Internally, valerianate of zinc and asafœtida, quinine, arsenic, belladonna, and bromide of potassium, are of most value. If the symptoms are chiefly bronchial, they may be treated, like ordinary asthma, by nitre-paper or stramonium cigarettes.

EPISTAXIS.

Epistaxis, or bleeding from the nose, may depend upon local or general conditions. Among the former are acute catarrhal, and tubercular and syphilitic lesions: and traumatic lesions, such as blows and picking. Epistaxis is not uncommon in childhood and early youth from causes that are not always obvious, unless it be simply delicacy of tissue; it is less common in middle age, but is again frequent in elderly people, whose vessels are beginning to degenerate. It is thus related to atheroma. It occurs also at all ages in connection with Bright's disease, cirrhosis of the liver, cardiac valvular disease, the various diseases of the blood, such as scurvy, purpura, and different forms of anæmia, and some infectious diseases, such as enteric and relapsing fevers.

The bleeding is often from the anterior part of the septum; if blood flows from the posterior nares it may trickle down the fauces into the stomach, and be subsequently vomited or passed per rectum: or it may cause cough and give rise to a suspicion of hæmoptysis. A pre-existing headache is sometimes relieved by a moderate bleeding.

Epistaxis, even if abundant, usually ceases of itself, but it may recur so frequently, and thus cause so much anæmia, that treatment becomes essential. It may be stopped by keeping the patient in a sitting posture, raising the arms above the head, and applying an ice-bag to the back of the neck. If this is not sufficient the anterior or posterior nares should be plugged, and ergot, gallic acid, or other hæmostatic, may be given internally, or ergotin subcutaneously.

DISEASES OF THE LARYNX.

LARYNGITIS.

Laryngitis, or inflammation of the larynx, may be acute or chronic, and arises from a number of causes. Amongst these are—exposure to wet and cold, and the ordinary conditions of catarrhal inflammation; contact with irritating vapours, air charged with dust or other minute particles, or scalding water; the impaction of foreign bodies, or direct injury in other ways; extension of inflammation from surrounding parts, the pharynx, the bronchi and trachea, or the tissues outside; the growth of tubercle, cancer, and syphilitic gummata; the circulation of poisons in the blood, such as those of diphtheria and measles, and finally Bright's disease. The results differ somewhat according to the cause, and one can readily distinguish a catarrhal laryngitis, an œdematous laryngitis, the membranous laryngitis of diphtheria, and the laryngitis of phthisis and of syphilis.

ACUTE CATARRHAL LARYNGITIS.

Ætiology.—This is mostly the result of exposure to cold air, but also arises from irritating vapours, dusty air, the entrance of foreign bodies, inflammation spreading from the pharynx or bronchi, and as one of the effects of the poison of measles. Some people are liable to it whenever they get a severe nasal or bronchial catarrh.

Morbid Anatomy.—The disease consists of swelling and increased vascularity of the mucous membrane of the larynx, with the secretion of more or less mucus, or in later stages muco-pus. Occasionally slight abrasions of the epithelium occur, and less frequently hæmorrhage takes place into the tissue of the mucous membrane or on the surface. In very severe cases there is œdema of the submucous tissues. As a result of the inflammation of the overlying structures certain changes take place in the neuro-muscular apparatus of the larynx, especially paralysis of the thyro-arytenoid muscles (internal tensors).

Symptoms.—There is at first soreness or dryness of the throat, and the voice becomes hoarse or entirely lost. There is occasional irritating cough, shrill, husky, or toneless, with expectoration from time to time of small plugs of mucus. Respiration is generally but little affected, but there may, in exceptional cases, be some stridor, or mucous râles, produced in the larynx; and in children dyspnœa is much more often a marked symptom. Fever may be slight or none. On examination with the laryngoscope the larynx is seen to be reddened, either generally or in patches, the posterior ends of the vocal cords, the inter-arytenoid space, and the ventricular bands being most frequently affected. In consequence of the thyro-arytenoid paralysis, the cords fail to meet in the middle, leaving a fusiform opening, and to this, as well as to swelling of the inter-arytenoid fold, the loss of voice is to be attributed.

Children are liable to a form of acute laryngitis (*spurious croup, laryngitis stridulosa*), which is characterised by the sudden development of suffocative symptoms, frequently in the middle of the night. A child, who has gone to bed apparently well, wakes up suddenly in terror, with severe dyspnœa, and a barking or husky cough, followed by loud and prolonged crowing inspiration. The voice is husky and feeble, and the features are congested; if the condition continues, the face may become pale and livid, and suffocation seems imminent. Usually, however, in a short time the symptoms become less severe, and the child falls asleep. Either on the same night, after a few hours' sleep, or on subsequent nights, the same attacks of threatening suffocation with croupy inspiration may take place. In association with these attacks, there is more fever (white tongue, flushed face, hot skin, &c.) than commonly occurs in catarrhal laryngitis of adults. The attacks have been attributed to muscular spasm set up by laryngitis; but Mackenzie attributes them to the inspiration of mucus in the narrow glottis of the child during sleep. These symptoms are apt to recur in the same individual whenever he catches cold; they are, however, rarely fatal.

The **Prognosis** is mostly favourable; acute laryngitis generally subsides in the course of a few days.

The **Diagnosis** is generally simple, especially in adults; diphtheria is more severe, and may be accompanied by membrane on the fauces, by the expectoration of membrane, or by albuminuria.

Treatment.—For acute laryngitis the patient should be placed in a uniformly warm atmosphere, and use steam inhalations from a suitable inhaler frequently. This may be charged with tr. benzoin co. (ʒj. to a pint of water), or succus conii (ʒij., with sodii carb. exsic. 20 gr.), or lupulin (ʒss.); or the air may be kept constantly moist by the use of a bronchitis-kettle. Demulcent liquids should be drunk freely, or small pieces of ice may be sucked. The irritation of cough should be allayed by opiates. The diet or regimen usual in febrile affections will of course be followed. It is of great importance that the patient should abstain as much as possible from using the voice. Local applications by the laryngeal brush seem not to be advisable till the later stages, when astringent solutions may be used (*see* p. 395).

For spurious croup an emetic is often useful, such as sulphate of zinc (5 to 10 grains), or ipecacuanha (2 to 5 grains of powder, or a drachm of the wine every ten minutes, till vomiting is produced). In addition, hot flannels or a hot sponge should be applied to the throat. In the intervals, the laryngitis is to be treated by a moist warm atmosphere (steam-kettle) and small doses of bromides or chloral.

Œdematous Laryngitis.

Ætiology.—This occurs specially in young adults, and in males more often than in females. It is sometimes caused by exposure to cold, and not infrequently, according to Mackenzie, is brought on by septic influences among hospital nurses, students, and others. It may be a result of the administration of iodide of potassium. Inflammations of the pharynx, probably of erysipelatous nature, and sometimes diphtheria, disease of the cartilages and perichondrium, such as occur in enteric fever, syphilis, and phthisis, cellulitis of the neck spreading from aneurysm and tumour in the chest, or that known as *angina Ludovici*, and the contact of boiling water, are among its causes; and it also occurs in Bright's disease.

Morbid Anatomy.—It consists of an effusion of inflammatory serum into the submucous tissue, and the serum contains more or less leucocytes, so that it may be sero-purulent; or actual pus is diffused through the tissue.

Symptoms.—These are often very rapidly developed, and within a few hours the patient may be in imminent danger from the obstruction to respiration produced by the swelling. Besides the dyspnœa, which is the prominent symptom, dysphagia is also experienced, the voice becomes hoarse, feeble, or disappears entirely, and there is more or less stridor in respiration. The laryngoscope reveals enormous swelling of the epiglottis, which forms a thick semicircular fold, or shows its two lateral halves much swollen and pressed together; in colour it is bright red. The swelling also affects the ary-epiglottic folds and the ventricular bands, but rarely the vocal cords themselves. The swollen epiglottis can be also felt by the finger, or even seen on depressing the tongue with a spatula. Care must be taken not to set up a spasm of the larynx.

A *subglottic* œdema is also described, but in Mackenzie's experience this is of a chronic nature.

The **Diagnosis** rests on actual inspection with the laryngoscope; in its absence, examination of the epiglottis with the finger may help.

The **Prognosis** in cases of extensive œdema is a serious one; in those which arise from blood-poisoning the latter may be fatal even if the dyspnœa is relieved.

Treatment.—The object should be to remove the laryngeal obstruction as soon as possible. Local bleeding by means of leeches, inhalation of tannin spray (Trousseau), sucking ice, potassium bromide in 10 or 20 grain doses, and the subcutaneous injection of pilocarpin nitrate may cause the œdema to subside; but if the dyspnœa is urgent the swollen epiglottis should be scarified by the laryngeal lancet, or by a curved bistoury, covered nearly to the point with adhesive plaster, and the throat should be gargled with warm water after the operation. If this fails, intubation or tracheotomy must be performed. Quinine and iron should be given internally.

MEMBRANOUS LARYNGITIS.

Ætiology.—The most common cause of membranous laryngitis is diphtheria, and it has been already shown (p. 110) that this disease, beginning in the fauces, frequently spreads to the larynx and trachea, and there produces the characteristic membrane, with dangerous suffocative symptoms. But quite independently of faucial inflammation, a membranous laryngitis occurs, of which the laryngeal and tracheal symptoms are precisely similar to those occurring in undoubted diphtheria, and which is undoubtedly, in the majority of cases, diphtheritic.

It is of interest to note that these primary laryngeal cases are more common in children than in adults, and that they are less often accompanied by albuminuria, or followed by paralysis, than those in which the throat first suffers.

A membranous laryngitis also occurs occasionally in connection with other specific fevers, such as scarlatina or measles; and it may certainly be produced by traumatic causes or local irritants, such as chemical vapours, boiling water, or impacted foreign bodies.

Symptoms.—The local symptoms will be like those already described under diphtheria (*see* p. 109); but the toxic symptoms of an infectious disease will be absent or little pronounced, when the cause is more distinctly traumatic.

Diagnosis.—In the majority of children taken with dyspnœa, ringing or "croupy" cough, and retraction of the chest, without apparent cause, and threatened with suffocation in from one to three or four days, membranous laryngitis is present; but it is generally impossible to carry out an examination with the laryngoscope, and the first proof of the presence of membrane may be provided at or after the operation for tracheotomy. It is distinguished from laryngitis stridulosa (p. 391), by the more gradual development, and more uniform progress of the dyspnœa.

Treatment.—Membranous laryngitis may be treated as shown under diphtheria, both when it is due to this disease, and when it is associated with scarlet fever, measles, or other infectious illness. The antitoxin of diphtheria should be used in the first case.

Chronic Catarrhal Laryngitis.

Ætiology.—This is often the result of acute laryngitis, especially when the latter is not properly treated with complete rest of voice. It is seen in those who use the voice continuously for long periods, and with much exertion, like costermongers, schoolmasters, and clergymen. It may extend from the pharynx, and is thus often induced by the excessive use of alcohol or tobacco.

Symptoms.—There is hoarseness of voice, accompanied by dryness, irritation of the throat, and tickling on prolonged use of the larynx; or there may be loss of voice. The cough is frequent, but there may be only a little hawking-up; and the expectoration, which is mostly viscid mucus, is never abundant. The symptoms are often most marked after an interval of rest, and disappear during the effort of talking, until after a time fatigue again ensues. Dyspnœa is generally absent. With the laryngoscope more or less congestion of the mucous membrane is seen to be present, and this may be diffused or unequally distributed, and mucus is here and there adherent to the surface. In old cases the mucous and submucous tissues become swollen and thickened, especially over the epiglottis, interarytenoid fold and ventricular bands, and the vocal cords may become granular. The mobility of the vocal cords is impaired, partly by a paralysis of some of the muscles of the larynx, which, according to Ziemssen, is more often unilateral than bilateral; partly by the thickening of the mucous membrane. Indeed, the swollen interarytenoid fold may project between the cords on phonation so as to hinder their closure. Erosions, or superficial ulcerations, are often present, especially on the cartilaginous part of the vocal cords, and between the arytenoid cartilages.

Diagnosis.—This must be made from the history and the laryngoscopic appearances, in which the thickening has to be distinguished from the transparent swelling of œdema, and the dull-red swellings of tubercular laryngitis. In cases of long standing the possibility of its connection with phthisis must be carefully inquired into, and in older patients malignant disease may cause a chronic thickening, which is likely to affect one cord only, and to impair its movement.

Treatment.—The condition is very troublesome, and requires persistent treatment. The patient should protect himself from cold and exposure by suitable clothing, as well as by confinement to the house in bad weather; and he should talk as little as possible. Change of residence to a mild and equable climate may be also beneficial. Astringents should be applied by means of the laryngeal brush. For this purpose the following solutions may be used: Cupri sulph., 10 grains to an ounce of water or glycerin; zinci chlorid., 30 grains; argent. nit., 10 to 30, or even 90 grains; zinci sulph., 10 grains; alum, 30 grains. One of these should be employed daily for seven days, on alternate days during the next two weeks, and so on with gradually decreasing frequency. The spray inhalations are also useful, such as zinci chlorid., 2 grains to an ounce of water, or iron-alum, 3 grains; and these may be continued during convalescence. For cases with excessive secretion Mackenzie recommends the local application of turpentine; and carbolic acid (ʒss.-ʒj. to glycerin ℥j.) for cases with long-standing hyperæmia and diminished secretion. The paralysis of chronic laryngitis requires to be treated by electricity applied internally.

A *subglottic chronic laryngitis* has been described, in which the mucous membrane below the vocal cord becomes much thickened and hypertrophied, so as to be easily visible like a second true cord. Its exact nature is uncertain, but Mackenzie has found it result from disease of the cricoid and arytenoid cartilages. The prognosis is very unfavourable, and active treatment is required of the same kind as that employed in ordinary chronic laryngitis to prevent hypertrophy. In late stages bougies may be required to dilate the glottis, and tracheotomy has been several times found necessary.

Glandular laryngitis, in which the racemose glands are chiefly affected, is generally associated with follicular laryngitis. The symptoms are those of a mild chronic laryngitis, and require astringent treatment. Nitrate of silver solution, 20 grains to an ounce of water, has been especially recommended.

LARYNGEAL PERICHONDRITIS.

This variety of deep inflammation of the larynx requires a few words. It is mostly the result of phthisis, carcinoma, syphilis, or enteric fever affecting the larynx, or of simple chronic laryngitis. It may also arise from traumatic causes, such as direct injury by a blow or from cut throat, an impacted foreign body, the pressure of the larynx against the spinal column in patients confined to the recumbent position, or the frequent passage of bougies down the œsophagus. As a result of the inflammation the perichondrium becomes thickened, pus forms in its fibres and collects between it and the cartilage, which, separated from its nutritive supply, becomes necrosed. The structures superficial to the perichondrium also become inflamed, œdematous, and purulent; thus an abscess is formed, which contains the dead fragment of cartilage. This sequestrum may remain for months or years after the opening of the abscess: on its removal the parts will contract and cause deformity and stenosis of the larynx.

Symptoms.—Dull, aching pain, tenderness on manipulation, with difficulty of swallowing, and hoarseness of voice or aphonia, are the usual symptoms, varying somewhat according to the cartilage affected, and often very much masked in secondary cases; thus in typhoid fever, loss of voice may be the only symptom suggesting an inquiry into the condition of the larynx. If the membrane on the outer side of the thyroid is affected, there will be swelling in the neck and formation of an abscess. If the inner surface of the thyroid, or the cricoid or arytenoid cartilage, is affected, there is œdematous infiltration of the interior of the larynx corresponding to it, which will be visible with the laryngoscope. The mobility of the vocal cords may be lessened by paralysis of the posterior crico-arytenoids when the cricoid cartilage is diseased, and by direct implication of the cord when one arytenoid is affected.

The **Prognosis** is unfavourable; when the primary cause, as typhoid or syphilis, is not in itself fatal, troublesome contractions of the glottis ensue after discharge of the cartilage; and pneumonia and gangrene of the lung have occurred from insufflation of the offensive secretion.

Treatment.—In acute cases, the inflammation may be reduced by the application of leeches or an ice-bag to the surface. If an abscess forms it must be incised. Often, however, tracheotomy is required, and before the tube can be dispensed with, the contraction of the glottis has to be treated perseveringly by dilatation or other operative measures.

TUBERCLE OF THE LARYNX.

It is well known that amongst patients suffering from phthisis a considerable portion have a laryngeal affection, which has been described as *laryngeal phthisis*. Different opinions have been held as to its relation to the pulmonary disease, but two facts are now well established: one that the disease of the larynx is always preceded by disease of the lungs; the other that, in the large majority of cases at least, the disease is due to the deposit of tubercles in the laryngeal tissues. The tubercles occur as minute collections of cells in the mucous or submucous tissues, forming, perhaps, slight prominences on the surface, leading in time to more or less, often considerable, œdema of the surrounding parts, and later to ulceration. Extending more deeply in severe cases, the inflammatory process leads to perichondritis and necrosis of the cartilages. The most frequent seats of the deposit are the mucous membrane covering the epiglottis and the arytenoid cartilages, the ary-epiglottic folds, the ventricular bands, and the vocal cords. Paralysis of the vocal cords may be simultaneously caused by thickening of the right pleura involving the right recurrent laryngeal nerve, or tuberculous bronchial glands pressing upon one or both.

The **Symptoms** are those of chronic laryngitis, and present nothing characteristic. In cases of ordinary severity, hoarseness of voice, frequent husky cough, and pain on swallowing, are the prominent symptoms. Sometimes in early stages the voice may be lost entirely from functional failure, in later stages, from paralysis of a vocal cord, or extensive destruction; and swallowing may be not only painful but difficult on account of swelling of the tissues, or from their destruction preventing perfect closure of the larynx. The cough is occasionally severe and paroxysmal, and expectoration is variable, depending rather on the condition of the lungs than on that of the larynx. In a small number of cases, considerable obstruction to respiration arises. In early stages the laryngoscope shows pallor of the mucous membrane; and a decided anæmia of the larynx occurs quite early in many cases of phthisis. When œdema takes place it often assumes a characteristic appearance, the ary-epiglottic folds on one or both sides being swollen up into a pale globular or pyriform tumour—the base backwards, the point forwards; and when both are affected the swellings coalesce in the middle line. The epiglottis is often œdematous and thickened, forming a turban-shaped swelling; and the same thickening may affect the ventricular bands, which are, however, often concealed. Subsequently, numerous ulcers form upon the swollen tissues. The vocal cords, though not much thickened, also become the seat of ulcers, which occupy chiefly the posterior halves.

Diagnosis.—This must be made partly from the laryngoscopic appearances, partly from the condition of the lungs. Many cases occur in patients with obvious phthisis. The pyriform swellings of the ary-epiglottic fold are characteristic of the condition; but when they are absent there may be difficulty in distinguishing it from *chronic catarrhal laryngitis* and from *syphilitic* disease. In the former there are less swelling and more congestion than in tubercular laryngitis; in syphilis the ulcers are generally larger and deeper, situate upon a more inflamed base, and solitary; the thickening is more irregular, and the disease often unilateral. Cancerous ulceration is sometimes difficult to distinguish from that of tubercle; cancer is often unilateral, and occurs in older patients.

Prognosis.—This is naturally bad, and permanent arrest of the symptoms is quite rare.

Treatment.—In early stages relief is obtained by the use of mineral astringents, as in chronic laryngitis. Perchloride and sulphate of iron have been especially recommended. Inhalations of the vapours of compound tincture of benzoin (ʒss. in a jugful of boiling water) or of lupulinum (ʒss.) are of value. Where the cough is troublesome the greatest benefit is derived from insufflations. Morphine acetate ($\frac{1}{8}$ gr. to $\frac{1}{2}$ gr.), with $\frac{1}{2}$ gr. of starch, can be sprinkled on the larynx by means of the laryngeal insufflator, a tube with a curved nozzle, which is introduced into the back of the mouth, over the larynx, with the aid of the laryngoscope. One or two grains of a mixture of morphine acetate, 1 part, boric acid and iodoform 2 parts each, and starch 7 parts, may also be used. A cocain spray (10 per cent.), and menthol pastilles are also useful.

Where there is ulceration some success has attended the repeated use of an aqueous solution of lactic acid (20 to 80 per cent.). which should be applied firmly by means of a pledget of lint; and the surface may in some cases be with advantage scraped by a *curette*, both to remove diseased tissue and to help the action of the acid. Rosenberg advocates the injection of a 20 per cent. solution of menthol in olive oil. Fifteen minims are used at the first sitting, and larger quantities afterwards. Fluids thickened with arrowroot, cornflour, or isinglass, are best given where swallowing is difficult from food entering the larynx, or, in extreme cases, an œsophageal tube may be necessary. Œdema may have to be treated with scarification, and tracheotomy may be required in urgent dyspnœa. The general treatment suitable to cases of phthisis must at the same time be continued.

SYPHILIS OF THE LARYNX.

The larynx appears mostly to be affected in the later stages of syphilis, often several years after infection; but occasionally earlier manifestations occur. Amongst these are chronic hyperæmia, superficial ulcerations, and condylomata or mucous patches: the last are very rare. In the later stages of the disease, gummata and deep ulcerations occur. The former are round elevations of the same colour as the rest of the larynx, from the size of a pin's head up to that of a pea, generally single and situate on the posterior wall of the larynx, the epiglottis, ventricular bands, or ary-epiglottic folds. They may ulcerate deeply or become absorbed. The ulcers of late syphilis, though in themselves not unlike tubercular ulcers, solitary, or only two in number, and often confined to one side of the larynx, are generally deeper, and surrounded with more inflammatory redness. They also develop very rapidly in a few days. Laryngeal œdema, and perichondritis with laryngeal necrosis, occasionally result, and the cicatrisation of ulcers frequently leads to the union of parts of the larynx to each other, or to the pharynx, so that serious distortions of the larynx or contractions of the glottis ensue. Thus the cords may be united by a web, or the epiglottis may be fixed to the pharynx.

Symptoms.—These are not distinctive, and vary much, according to the severity of the lesion. They are hoarseness or loss of voice, occasionally cough in earlier stages, and more or less dyspnœa in later stages. Swallowing is often painful, though the absence of pain when not swallowing is remarkable. Muco-purulent expectoration with blood may accompany extensive ulceration, and in a few cases free hæmorrhage has taken place.

Hereditary syphilis of the larynx in children has been seen by

Mackenzie in the form of ulcerations of the epiglottis. German writers have recorded instances in infants.

Diagnosis.—Syphilis of the larynx may be confounded not only with tubercle, but also with cancer. In this last the ulcer is generally preceded and accompanied by growth, in the form of nodular excrescences. The ulcers of cancer may be very large, and the surrounding tissue is inflamed. The hasty diagnosis of syphilitic laryngitis from the history alone, without a laryngoscopic examination, is to be deprecated. Dyspnœa and stridor in such persons may arise from paralysis of the vocal cords, from aneurysm pressing on the trachea or laryngeal nerves, or from syphilitic stenosis of the trachea or one bronchus.

Prognosis is not specially unfavourable to life.

Treatment.—Syphilitic lesions yield to the vigorous use of mercury (especially by inunction) or of iodide of potassium. The resulting contractions may necessitate tracheotomy or intubation. The voice is then commonly lost, and generally a tube has to be worn for life; but attempts may be made to dilate the glottis mechanically, or to divide a web by the cutting forceps, or dilator, or by the electric cautery.

LUPUS OF THE LARYNX.

Lupus, of which a fuller description is given under Diseases of the Skin, rarely attacks the larynx; although the systematic examination of this part in persons with lupus of the skin has shown it to be often present when not in the least suspected.

It occurs as pale or dark-red nodules, or papillary outgrowths, isolated or grouped upon a hyperæmic base, appearing first and most often upon the epiglottis, then upon the ary-epiglottic folds, and posterior wall of the larynx, and least often upon the vocal cords. As they increase in size they cause a general thickening of the parts. Later on, ulceration takes place, and the ulcers are followed by scars, in which again fresh nodules may appear; but the cicatrisation rarely leads to high degrees of stenosis.

Symptoms.—There are some soreness, trouble in swallowing, and alteration in the voice, which becomes hoarse or is even lost. Dyspnœa may occur in late stages.

Treatment.—This has consisted chiefly of local applications of tincture of iodine, iodoform, nitrate of silver and lactic acid, and the use of the electric cautery, or of the curette (see Tubercle of the Larynx). Cod liver oil should be given internally.

TUMOURS OF THE LARYNX.

BENIGN TUMOURS.

These are papilloma, fibroma, mucous cyst, myxoma, angioma, and lipoma. The last three are quite rare, and occur especially in children.

Papillomata are the most common. They are frequently about the size of a pea, but may be as small as a mustard-seed, or in rare cases as large as a walnut. They are pink, whitish-grey, or red, have an uneven, or papillated, or warty surface, and grow mostly from a broad base. They are often multiple, and their usual seat is the vocal cord on one or both sides, or the angle between the cords; sometimes the ventricular bands or the epiglottis. They are liable to recur after removal, and in a few instances have been known to undergo epitheliomatous degeneration.

Fibromata, or fibrous polypi, are of slower growth, and show no tendency to recur. They are round or oval, sessile or pedunculated, with a smooth surface and bright red colour. Usually of hard consistence, they are more rarely soft, and contain a good deal of serous fluid in the meshes of the fibrous tissue. They are generally single, and arise from one of the vocal cords.

Mucous cysts arise commonly from the epiglottis, and are surrounded with an area of injected mucous membrane. They have dense walls, and are filled with thick, white, sebaceous material, or thinner yellowish or brown fluid.

The **Symptoms** of tumours depend upon their seat. If situate upon the vocal cords the voice is impaired or lost; and impairment of voice is the commonest symptom. If the tumour is sufficiently large, dyspnœa is present. Dysphagia from implication of the epiglottis, and cough, generally dry and hacking, also occur. In children the cough may be croupy. Pain appears to be rare.

The **Treatment** is removal by surgical operation, for the details of which the reader is referred to surgical works, or special treatises.

MALIGNANT TUMOURS.

These are mostly epithelioma, but scirrhus, encephaloid cancer, and sarcoma also occur. They are more frequent in men than in women, and appear commonly in advanced life. The posterior part of the cricoid cartilage and the vocal cords appear to be the most frequent seats of commencement of the growth (Semon); but it may begin in the epiglottis, or the ventricular bands, and it may involve the whole larynx, so that after a time it is impossible to say where it has begun. The appearance is at first not distinctive; it may be an infiltrating or warty growth, or a definite tumour. But it shortly ulcerates, vegetations spring up about the margins in epithelioma, and these ulcerating in their turn, the disease rapidly spreads. The surface is often covered with pus, or sanguineous muco-pus, and occasionally free hæmorrhage takes place. Œdematous laryngitis and perichondritis occur as complications. The larynx is, of course, affected sometimes by cancer spreading from the pharynx or the neck.

Symptoms.—Dyspnœa, dysphagia, and alteration of the voice occur as in other laryngeal affections. An important feature is *pain*, which is at first local, but subsequently radiates to the ear, orbit, or forehead. As ulceration proceeds, the breath becomes fœtid, and hæmorrhage may occur. The voice early becomes hoarse, but is rarely completely lost. Occasionally the sub-maxillary glands are implicated, and in rare cases the thyroid cartilage is distended by the growth within.

The **Diagnosis** is often difficult at first, but on the appearance of a tumour its features, the age of the patient, and the absence of a history of syphilis, or of long-continued chronic laryngitis, will afford strong evidence of its cancerous nature.

The **Prognosis** is unfavourable, and the only successful **Treatment** must be surgical. A sufficient number of operations for the removal of the growth have been successful to justify this procedure being considered whenever an early diagnosis can be made.

FOREIGN BODIES IN THE LARYNX.

There are a large number of foreign bodies which have at different times found their way into the larynx. Among these are peas, beans, buttons, coins, fragments of bone, shells, pebbles, artificial teeth, portions of solid food, and pieces of children's toys. The obstruction may be at once fatal, or it may be so slight as to be scarcely noticeable. Indeed, it should be remembered that it is not always known to patients or their friends that a foreign body has been introduced. Thus a child attending among my out-patients for phthisis, was one day left in a perambulator outside a shop, while its mother went in to purchase something; it was suddenly taken with "convulsions," became black in the face, and died. It was only on *post-mortem* examination that it was discovered that a pea had got into the larynx, where it lay completely occluding the glottis. Another interesting case was communicated by Mr. Golding-Bird and myself to the Clinical Society. A man came to the hospital for hoarseness and occasional cough. On examining the larynx I saw what appeared to be a white bony substance below the vocal cords. On telling him he appeared to have a piece of bone in his throat, he remembered that three months previously he had by accident swallowed a piece of bone in some sheep's-head broth, that it stuck in his throat and nearly choked him, but that his wife forced a passage with the gravy-spoon. He was relieved, and, though the laryngeal trouble followed, he had no idea but that he had properly swallowed the piece of bone, which proved, on removal by laryngotomy, to be three-quarters by five-eighths of an inch in size.

Impacted foreign bodies may give rise to secondary inflammation, œdema, or hæmorrhage, or to pain and spasm. Sometimes a change in their position may cause sudden death. Their position and relations should be accurately estimated, and they should be removed, if possible, by forceps through the upper opening of the glottis, even when they are below the cords. But they may require thyrotomy, or infra-thyroid laryngotomy.

PARALYSIS OF THE LARYNGEAL MUSCLES.

From the peculiar course of the recurrent laryngeal nerve—the chief motor nerve of the larynx—paralysis of these muscles has often a diagnostic importance beyond that of the trouble arising locally. But it may be caused by lesions not only of the laryngeal nerves, but also of the vagus above their origin, and of the medulla where the nuclei are situate. Thus laryngeal paralysis is a part of "bulbar paralysis," results from syphilis and tumours affecting the medulla oblongata, and occurs occasionally in association with locomotor ataxy and insular sclerosis. The vagus in the neck may be compressed by tumours and enlarged glands, or may be injured by wounds or operations. The recurrent laryngeal nerves are in danger in two situations—the thorax and the neck; and the left is the more liable to lesion from its curving round the arch of the aorta, whereas the right goes no lower than the subclavian artery. Either of them may be sometimes involved in the fibrous thickening at the apex of the lung in chronic phthisis, but the left is especially liable to be compressed by mediastinal tumours, by enlarged bronchial glands, and by aneurysm of the arch of the aorta. In the neck the two nerves ascending to the larynx lie between the trachea and the œsophagus, and may be involved together in cancer of the latter, or compressed by an enlarged thyroid body. Paralysis also occurs, as already stated, in catarrh, and from other local affections of the larynx; as a result of diphtheria, influenza, and other infectious diseases, chronic alcoholism, and poisoning by lead and arsenic; and, finally, as a purely functional failure, or part of hysteria. The more important forms of paralysis will now be described.

COMPLETE PARALYSIS OF THE VOCAL CORDS.

This is the result of lesions dividing or severely injuring the recurrent laryngeal nerve, or the vagus above; or of diseases of the medulla oblongata—*e.g.*, bulbar paralysis. Of the nerve-lesions, neuritis from alcohol and diphtheria must not be forgotten. The paralysis may be bilateral, which is very rare, or unilateral. When bilateral, the cords assume what is known as the *cadaveric position.* They are immovably fixed midway between the positions of *ab*duction and *ad*duction, each cord with a concave margin, leaving a fusiform interval between them, unaffected by inspiration, expiration, or attempts at phonation. The aperture is sufficiently wide for respiration, hence there is no dyspnœa; but phonation is impossible, as the cords cannot be approximated, so that the patient speaks in a whisper, and the acts of coughing and expectoration are imperfectly performed. Some stridor is produced on forced inspiration, probably from the vocal cords, ary-epiglottic folds and arytenoid cartilages being thrown into vibration.

When the paralysis is unilateral, the cord affected lies in the cadaveric position, but from the mobility of the sound cord the symptoms differ somewhat. Again, there is no dyspnœa. The voice may be entirely lost, but often it is produced by the healthy vocal cord being drawn completely over the median line till it comes into contact with the paralysed cord. The voice is then harsh, and may break into falsetto on an increase of the effort. The cough has often a peculiar brassy or clanging quality.

PARALYSIS OF THE ABDUCTORS.

Although the recurrent laryngeal nerves, supplying as they do all the muscles of the larynx except the crico-thyroid, must contain fibres for both *ad*ductors and *ab*ductors, it is a remarkable fact that coarse lesions of these nerves (compression by tumours or aneurysms) may result at first in paralysis of the *abductors* alone. Abductor paralysis also results from lesions of the medulla, where it may be supposed it sometimes depends on a separate affection of the nucleus of the abductor fibres; though it is to be noted that adductor paralysis alone never arises under such circumstances. This greater liability of the abductor fibres to suffer from lesions affecting the whole nerve or centres is not explained by anatomical arrangements, but corresponds to a difference in physiological properties. Pressure on the trunk of one vagus nerve may cause double abductor paralysis by reflex influence. Abductor paralysis is probably sometimes the result of a change primarily in the muscle; it is rarely the result of hysteria.

The first effect of the lesion is that the cord during respiration not being fully abducted remains in the cadaveric position, and allows ample space for the passage of air; after a time, however, the antagonist muscle, or adductor, contracts (*paralytic contracture*), and the cord is drawn into a position of adduction. Thus, in bilateral paralysis of the abductors the cords are seen to be permanently approximated in the middle line to within one-tenth of an inch of each other; on attempted phonation they meet completely; on inspiration they do not separate, but are even drawn a little closer together; on expiration they scarcely move, or only in the reverse sense to their slight movement in inspiration. The important symptom is *dyspnœa*, which results from the permanent narrowing of the glottis; this is generally accompanied by *stridor* on inspiration, which is worse on exertion, and often extremely loud during sleep. The voice is clear, or it may be a little hoarse. Coughing can be perfectly effected.

When only one cord is paralysed, the dyspnœa is less serious, and the stridor less marked; and they may only be present on exertion. The voice may be normal.

Diagnosis.—Abductor paralysis may be confounded with spasm of the abductors, with ankylosis of the arytenoids in the position of adduction, and with perverted action of the cords in which they move inwards instead of outwards during inspiration. It is important to remember that the lesion which causes unilateral or bilateral abductor paralysis may at the same time cause narrowing of the trachea, by pressure (aneurysm, tumour) or by cicatrix (syphilis); and the dyspnœa and stridor due to the latter may be wrongly attributed to the former. Tracheal stenosis usually causes expiratory as well as inspiratory stridor.

The **Prognosis** is generally serious. Except when hysteria or syphilis is the cause, there is little hope of recovery; and while some cases may last for years without any change, there is a constant liability to death from suffocation. If the adductors are subsequently paralysed the obstruction is diminished and aphonia develops. Death may arise from the primary lesion, such as œsophageal cancer, or double aneurysm. In long-standing cases the posterior crico-arytenoid muscles become completely atrophied.

Treatment.—Where the cause of double paralysis is central, or there is a possibility of syphilis being the cause, iodide of potassium and perchloride of mercury may be given internally, and the tone of the muscles improved by faradism and galvanism applied by laryngeal electrodes; the subcutaneous injection of sulphate of strychnia ($\frac{1}{30}$ gr.) has also been recommended. But if no improvement takes place in a few weeks, and if dyspnœa is constant, or night attacks take place, tracheotomy should be performed, and the tube worn constantly.

In unilateral paralysis the risk of asphyxia is much less, and the treatment may be directed mainly to the cause.

PARALYSIS OF THE ADDUCTORS.

This is mostly the result of functional disease or hysteria, and rarely occurs alone from structural lesions. Two sets of adductors are described—the lateral adductors, or *crico-arytænoidei laterales*, and the central adductor, or *arytænoideus proprius;* but the inner fibres of the *thyro-arytænoidei*, or internal tensors, also act as adductors of the anterior portions of the vocal cords. In the most common form of adductor paralysis these are all affected. When examined with the laryngoscope, the glottis is seen to be widely open; on attempts to speak, the cords scarcely move at all, but remain still at the sides of the larynx. As the cords cannot be approximated, the patient speaks only in a whisper, no laryngeal voice being produced, though sometimes, with an effort, a momentary contact of the cords may be effected. Coughing, in which the cords are brought together by involuntary reflex action, is generally perfect; and from the open condition of the glottis there is no dyspnœa. This constitutes *functional* or *hysterical aphonia*, which is, however, often started by slight catarrh of the larynx, or by sore throat, or by other local trouble; but more generally, like other hysterical conditions, by mental disturbances or bodily illness.

Sometimes the adductor paralysis is less extensive; the internal tensors may be alone affected, so as to produce want of contact of the cords on attempted phonation, each cord presenting a concave margin to its fellow in its anterior half. And sometimes the central adductor is paralysed, in which case the anterior portions of the cords come into contact, and a triangular space is left open behind, between the arytenoid cartilages. These last two forms are not uncommon in the course of catarrhal laryngitis. They may occur together, producing defective closure in front and behind, while the processus vocales are in contact. In these cases the loss of voice is not so complete as in that first described.

The **Diagnosis** of these conditions is easily made with the laryngoscope. Even without this, the voicelessness of the patient, the absence of dyspnœa, cough, and expectoration, and the power to cough at will, are sufficiently distinctive.

The **Prognosis** is favourable, and cases of many years' duration may be at length cured.

Treatment.—Functional aphonia must be treated by faradisation. Slight cases will often be cured by the application of a current to the neck, an electrode being placed on either side of the larynx. But long-standing cases require endo-laryngeal faradisation. One electrode should consist of a metal plate placed on the neck in front of the larynx, and retained by a band round the neck. The other is the laryngeal electrode, which must be introduced into the larynx with the aid of the mirror. The instrument is so made that the current passes to the free extremity (touching the larynx) only when a key in the handle is depressed by the operator. It is advisable in all cases to give a powerful shock even at first. Often the patient at once cries out, and recovers the use of the voice from that time. In other cases the voice is recovered for twenty-four hours or more, and is lost again till the muscles are again submitted to faradisation. Stimulating applications, inhalations, and sprays are also useful as adjuncts. The health of the patient should be maintained by tonics, and nervous failings should be met by the requisite moral treatment. Paralysis occurring in the course of laryngitis will generally spontaneously recover at the same time.

PARALYSIS OF THE EXTERNAL TENSORS.

The crico-thyroid muscles may be paralysed at one or both sides, but this is not a common occurrence. It arises from cold, or from prolonged use of the voice. The voice becomes gruff, or may be entirely lost. With the laryngoscope, the cords are seen to be applied to each other in a wavy line, instead of being perfectly straight and parallel. This condition should arise from lesions of the superior laryngeal nerve. Mackenzie records a case due to pressure of an inflamed gland on this nerve, but has chiefly known these muscles paralysed in connection with diphtheria.

Paralysis of the thyro-epiglottic and ary-epiglottic muscles, which depress the epiglottis, occurs also from lesion of the superior laryngeal nerve, and is accompanied by anæsthesia.

SPASM OF THE GLOTTIS.

In this affection the adductors are spasmodically contracted, and complete closure of the glottis takes place, preventing the entrance of air, and producing asphyxia, or even death. It may occur at all ages, but is especially frequent in infants, in the form now to be described.

LARYNGISMUS STRIDULUS.

(*Spasmodic Croup. Child-crowing.*)

This occurs between the ages of three months and two years, and is more common in boys than in girls. It is promoted by imperfect hygienic conditions, and is more frequent among the poor, and in those children that are hand-fed, or are nursed by sickly and half-starved mothers. Rickets, which occur under simila hygienic conditions, is observed in a large proportion (75 pe cent.) of the cases The predisposition being present, a numbe of causes may excite a spasm, such as crying, sucking, quick movements, milk getting down the larynx, indigestible food in the stomach, the irritation of dentition, and the presence of post-nasal adenoids; but the attacks often occur without any such obvious antecedent. The child may be in fairly good health, or may suffer from the symptoms common in rickets, when it is noticed to make a slight crowing sound occasionally. This may be repeated at intervals without giving rise to any alarm, but gradually becomes more frequent, and then is liable to be induced by the exciting causes above mentioned; however, it often occurs during sleep, and, on the whole, more during the night than in the day-time. After a while the interruption to respiration, at first only indicated by the crowing, becomes more marked. Breathing ceases, the chest is fixed, the face becomes pale and livid, the head is thrown back, and the facial muscles are slightly twitched. In a short time the spasm yields, and the air enters with a loud crowing noise through the still imperfectly opened glottis; and the child in a few minutes more may return to its playthings. In the severest cases the glottic spasm is accompanied by convulsive rigidity of the hands and feet, known as *carpopedal contractions:* the fingers are bent into the hand, the thumb within the fingers, and the hand is flexed on the wrist; the legs are extended, the feet bent on the legs, the soles turned inwards, and the great toe widely separated from the others. General convulsions may be added to these. Occasionally, death takes place during a fit, from complete stoppage of the respiration; and as the crowing is really the signal that the spasm is relaxing, it will be seen that in the fatal cases death may occur quite silently.

Many opinions have been advanced and discredited as to the causation of spasm of the glottis in children. Semon points out that bilateral cortical centres for the adduction of the vocal cords in phonation have been shown to exist (Krause, Semon and Horsley), and it is probable that their defective nutrition and consequent irritability may have a large share in causing the symptoms of this disorder.

Diagnosis.—The symptoms are very characteristic, and not easily confounded with any other. The absence of fever, the shortness of the attack, and the complete intermission between the attacks, distinguish it from laryngitis. A foreign body might cause symptoms like it, as shown by the case I have recorded (p. 402).

Prognosis.—This is, on the whole, favourable. The child may, indeed, die in a severe attack, and friends should be instructed what to do before a doctor can arrive. But if fatal results can be warded off, the disease in time subsides, or yields to treatment and the improved condition of the child's health.

Treatment.—This has to be considered in relation to the general health of the patient, and the occurrence of the attacks. The child must be put immediately under the best possible hygienic conditions: fresh air, well-ventilated rooms, and improvements in its food, where this is insufficient or unsuitable (*see* Rickets). Medicinally, cod-liver oil, or cod-liver oil with malt extract, is of great value, and the bromide of potassium or ammonium may be given three times daily, in doses of 2 to 5 grains, according to the age of the child. If the attacks are slight, sponging the child from head to foot two or three times daily with cold or tepid water, according to the season, often quickly stops them. The bowels should be attended to. In the more severe fits, the head should be raised, the surface of the body and face slapped with a towel dipped in cold water, and ammonia or acetic acid held to the nostrils. Or the body may be immersed in warm water, and cold water poured over the head and face. The administration, when the child can swallow, of tincture of castor, or of musk (gr. 1½ with sugar and gum in a teaspoonful of water), tickling the fauces, a few whiffs of chloroform, and enemata of 20 to 30 minims of tr. asafœtidæ, have also been recommended. Even after apparent death, artificial respiration will sometimes restore the patient.

Spasm of the Glottis in Adults.

This occurs more frequently in connection with laryngitis, œdema of the larynx, paralytic conditions, or the presence of foreign bodies; it may also be a danger in epilepsy, tetanus, hydrophobia, or chorea. The entrance of saliva or small particles of food or drink into the larynx may cause most dangerous spasm; and a certain amount is often induced by the application of medicated solutions to the mucous membrane of the larynx. Apart from these causes, it is most often the result of hysteria.

Treatment. — Inhalations of chloroform, vapor coninæ, stramonium, or datura tatula should be employed, if they can be obtained in time; otherwise tracheotomy may be necessary.

CHRONIC INFANTILE STRIDOR.

Infants are occasionally the subjects of a laryngeal disorder, in which the breathing is accompanied by a peculiar croaking sound. This is generally first heard soon after birth, is continuous for long periods, perhaps all day and night, but may be absent for a few hours at a time. The croaking takes place with inspiration, and is either a rough rhonchal sound, or more clear and musical; expiration is either quite normal, or rattling, as if from accumulated mucus; the cough and cry are, as a rule, normal. There may be a little sucking-in of intercostal spaces, but there is rarely any lividity. In some cases the noise is constant during sleep, in others it is absent; it is generally worse when the child is lively or excited. It subsides as the child grows older, but I have known it still present at two and a half years. The child appears to be in other respects quite healthy. Lees examined the larynx in one of these children who died of diphtheria, and found the epiglottis folded on itself, and the ary-epiglottic folds in contact. The same condition has been seen during life in a number of cases examined by Drs. Sutherland and Lack.

The obstruction caused by the deformity diminishes as the parts develop. No direct treatment is of any avail. Tracheotomy might be necessary in the rare event of life being threatened by asphyxia.

ANÆSTHESIA OF THE LARYNX.

This occurs in diphtheria, and in bulbar paralysis. It is recognised by the insensibility of the laryngeal mucous membrane when touched with a probe, introduced with the help of the laryngoscope. It is often accompanied by dysphagia from particles of food entering the larynx. This is, according to Mackenzie, from paralysis of muscles supplied by the superior laryngeal nerve, and not because of the insensitiveness of the mucous membrane. The muscles which fail, under these circumstances, are those which depress the epiglottis and close the upper aperture of the glottis during swallowing. Anæsthesia from diphtheria generally recovers; prognosis is generally bad in progressive bulbar paralysis.

The **Treatment** should be by galvanic and faradic applications. Strychnia may be given internally; and feeding with the œsophageal tube may be necessary if dysphagia is serious.

DISEASES OF THE TRACHEA.

TRACHEITIS.

Inflammation of the trachea arises from circumstances similar to those producing laryngitis. Acute catarrhal tracheitis frequently accompanies laryngitis and bronchitis, but is masked by the symptoms which they produce. Occasionally it exists alone. It then produces cough, often hacking, perhaps violent or paroxysmal, with some amount of expectoration. With the laryngoscope, the mucous membrane may be seen to be congested, and ulcers are sometimes observed. With the stethoscope, mucous râles may be heard in the trachea; but the swelling of the mucous membrane and the mucous accumulation are not generally sufficient to cause much dyspnœa. The patient requires treatment similar to that used in bronchitis—warm temperature and avoidance of exposure. Troublesome cough may be relieved by insufflations of morphia ($\frac{1}{16}$ to $\frac{1}{8}$ gr.) or bismuth nitrate ($\frac{1}{4}$ to $\frac{1}{8}$ gr.); and expectorants, such as squills and ipecacuanha, steam or benzoin inhalations, and the application of mustard to the upper part of the sternum, are of service.

The trachea is attacked by *diphtheria*, spreading from the larynx. Croup was at one time supposed to be mainly a tracheitis (*cynanche trachealis*), but it is now recognised that membranous inflammation of the trachea descends from the larynx.

Tubercle of the trachea occurs occasionally in association with tubercle of the larynx; ulceration follows the deposit of tubercle in the mucous membrane or submucous tissue. The ulcers are more common on the posterior wall, and usually measure from two to four millimetres, but may reach ten millimetres in diameter. The symptoms due to tracheal tubercle are generally masked by those to which simultaneous disease of the larynx or lung gives rise.

Syphilis, in its secondary and tertiary stages, also affects the trachea, producing in different cases, according to Mackenzie, congestion, condylomata (rarely), and superficial ulcers. The most important change, however, is stricture. The trachea is affected most often at its lower end, less commonly at its upper end; and the stricture may consist simply of a narrowing at one spot, or a considerable length of the trachea may be reduced in calibre. The mucous membrane is raised into bands and ridges, which have been regarded as cicatrices of former ulcers, possibly preceded by gummata; but German pathologists look upon the thickening as a direct result of the syphilis, and any ulceration which may occur as secondary. In late stages the cartilaginous rings have been exposed, necrosed, and expectorated or absorbed. The stricture can be sometimes seen below the glottis by means of the laryngoscope. For the symptoms, diagnosis, and treatment of stricture, *see* below, under Tracheal Obstruction.

NEW GROWTHS IN THE TRACHEA.

The trachea is remarkably little subject to primary new growths, whether benign or malignant. When present, they give rise to dyspnœa, and may be recognised, possibly, by the use of the laryngoscope. More frequently cancer of the œsophagus or of the mediastinum grows into the adjacent trachea, narrowing its channel and producing symptoms of stricture. Where it spreads from the œsophagus, it is preceded by dysphagia; but tracheal symptoms may be the first indication of cancer of the mediastinum. Another way in which tumours affect the trachea is by simply compressing it from outside.

As the chief symptoms in all these cases depend on the reduction of the calibre of the trachea, and as this may be due to other causes besides such tumours, it will be well to consider separately the pathology and clinical features of tracheal obstruction.

TRACHEAL OBSTRUCTION.

This arises in three ways: (1) From compression from without; (2) from changes in the walls of the trachea itself (stricture); (3) from foreign bodies within it.

Compression of the Trachea.—The most common causes are mediastinal new growths, aneurysm of the aorta or large vessels, enlargement of the thyroid body, and malignant tumours in the neck. Cancer of the œsophagus may also compress the trachea; but soon invades it, so that perforation takes place between the two tubes. Occasionally in children caseation and suppuration of the bronchial glands may lead to their enlargement, by which the trachea is compressed. The abscess may burst into the trachea, and pus or portions of caseous glands may be expectorated. A mediastinal abscess from any other cause, such as caries of the spine, may act in the same way. And, lastly, the trachea is sometimes pressed upon by the dilated left auricle in cases of mitral constriction.

Stricture.—The chief cause is syphilis, which has been already considered.

Foreign bodies are rarely retained in the trachea, but commonly fall into one or other bronchus, though they may be driven up and down the trachea by the respiratory currents.

Symptoms.—The most important are dyspnœa and stridulous breathing; they are often accompanied by cough, and the expectoration of thin frothy mucus. The voice is unaffected, or simply feeble from the obstruction weakening the current of expired air. The chest is resonant, but vesicular murmur is faint, or drowned by the noise of the stridor. Other symptoms accompanying tracheal stenosis are due to the lesion which causes it, and these may be at first entirely absent in a case of aortic aneurysm or deeply-seated mediastinal tumour.

Diagnosis.—This has to be made (1) between obstruction in the trachea and obstruction in the larynx; (2) between the different causes of tracheal obstruction.

The first may be determined at once by the laryngoscope, by which the absence of laryngeal disease may be proved, and even the presence of tracheal stricture, or of tumour or aneurysm compressing this tube, may be observed; but there are other points of distinction that are of value, especially as patients with severe dyspnœa do not always readily submit to laryngoscopic examination. One is the fact noticed by Gerhardt, that in laryngeal obstruction the larynx is moved extensively up and down in the neck during respiratory movements, whereas in tracheal obstruction it moves but slightly. In laryngeal obstruction the head is thrown back; in tracheal obstruction it is often bent forward. The stridulous breathing does not always offer points for distinction. Fagge says it is mainly inspiratory in tracheal as it is in laryngeal obstruction; but the expiratory stridor in the former is certainly louder than commonly occurs in double abductor paralysis, and this is a form of laryngeal stenosis from which tracheal obstruction has often to be diagnosed; for instance, where symptoms point to aneurysm or mediastinal growth, and the question is whether the dyspnœa is due to compression of the trachea, or of the recurrent laryngeal nerves. Under these circumstances, the growth may press both on the nerves and the trachea, and a double cause for dyspnœa exists, when even the laryngoscope will not help us to a knowledge of the condition of the trachea. Auscultation of the trachea is deceptive, as the loudest stridor is heard over the larynx even when the stenosis is in the trachea. This I have verified on more than one occasion. The point is of practical importance, because laryngeal obstruction may be relieved by tracheotomy, but tracheal obstruction rarely so; and it is desirable to spare the patient an operation of this nature when it can do no possible good.

The recognition of the cause of tracheal obstruction depends upon collateral symptoms. Any source of compression would probably involve other organs and thus cause dysphagia, obstruction of the veins of the head, neck, or arm, pressure on corresponding nerves, and dulness under the sternum, or at the uppe part of one or other chest. On the other hand, stricture due, a already stated, to syphilis must be free from such symptoms; bu an aneurysm of the aorta may compress the trachea without, a first, any other symptom by which it can be recognised. For th diagnosis of mediastinal growth from aneurysm, the reader i referred to Diseases of the Mediastinum.

Prognosis.—This is very unfavourable, the commoner cause being little amenable to treatment; but the rare cases of absces compressing the trachea may recover on the bursting of th abscess.

Treatment.—The indications are (1) to remove the cause, i possible; (2) to open the trachea below the obstruction where thi is in the upper part; and (3) to relieve symptoms and secondar results.

A thyroid tumour may be removed, and enlarged glands o growths in the neck; and abscesses, where accessible, may possibl be opened; but such opportunities are infrequent. If an aneurysm is diagnosed, the treatment for that condition should be employed and for obvious stricture, active anti-syphilitic treatment b means of mercury and potassium iodide. The latter drug shoul be employed in any case which does not present sufficient dat for a positive diagnosis as to the cause of the obstruction. In th case of a foreign body, tracheotomy should be performed, and the efforts to dislodge it should be made by inversion of the patient shaking, &c.

DISEASES OF THE BRONCHI.

BRONCHITIS.

Ætiology.—Bronchitis, or inflammation of the bronchi, occur at all ages, and may arise from a variety of causes, amongst whic the most frequent is exposure to cold or wet, or both combined Under such circumstances it may be associated with simultaneou inflammation of the larynx and nasal mucous membrane, o the inflammation may commence in the latter, and spread downwards to the bronchi. Another cause is contact of the bronchia mucous membrane with irritating vapours, as air carrying soli particles, such as dust, fog, or the air of mines and of certai manufactories. Bronchitis may also be set up by the presence o foreign bodies actually in the bronchial tubes; this is comparatively rare, but blood effused into the tubes may act in this wa and it constantly occurs as a result of the deposit of tubercle or cancer in the substance of the lung. Certain infectious diseases, already described, are frequently accompanied by bronchitis —namely, typhoid fever, measles, scarlet fever, diphtheria, influenza, and whooping-cough; and it often occurs in Bright's disease.

Among causes that may be called predisposing are age, habits, the general health, and preceding conditions of the lungs and heart. Bronchitis is especially prevalent amongst infants, young children, and elderly people; whereas young adults and the middle-aged are much less subject to it. Habits of luxury, confinement to warm rooms, and undue wrapping up, render the subject liable to contract bronchitis on comparatively slight exposure; and those in weakly health, or depressed from insufficient food, exhausting occupations, or bad sanitary conditions, easily acquire it. Such conditions are more common amongst the poor than amongst the wealthy. The pre-existence of heart disease, impeding the circulation in the lungs, and of former attacks of bronchitis—all the more if they have left behind them emphysema or dilated tubes—will tend to the ready development of the disease. Some other conditions of ætiological importance are those which involve constant exposure to the exciting causes, such as residence in towns, in cold, damp, and changeable climates, employment in mines, in wool and steel manufactures, and other such industries. Bronchitis is much more common in winter than in summer.

Pathology.—The mucous membrane is the part most affected, but in severe or prolonged cases the submucosa is involved, and, rarely, the cartilages of the bronchial tubes, and adjacent parts of the lungs. The first effect is increased vascularity and swelling of the mucous membrane, and after a short time a free secretion from the surface takes place. This catarrhal secretion is provided (according to Ziegler), partly by the blood-vessels, and partly by the epithelial cells and mucous glands in the large divisions. It consists chiefly of mucus, and contains leucocytes and shed epithelial cells. In later stages the secretion becomes more and more opaque from the presence of increasing numbers of leucocytes, extravasated, according to most writers, from the blood-vessels, but according to others (Socoleff, Hamilton), produced by germination from flat cells in contact with the swollen basement membrane after the superficial layers of the epithelium have been shed. The secretion may also contain cells in a state of fatty degeneration, or cells containing particles of soot or dirt derived from the inspired air. Sometimes the smallest tubes at the base of the lung are filled with thick green pus. If the superficial part of the base of the lung be sliced off, and the exposed section be squeezed, minute drops of pus will be found to ooze freely from the cut surface. The condition is one of *capillary bronchitis.*

If, instead of clearing up, the inflammatory process continues, so as to become chronic, the fibrous coats of the bronchi become thickened, and infiltrated with leucocytes; the muscular fibres become atrophied by pressure; and the cartilages and mucous glands disappear from the same cause. Ultimately, in many cases, the bronchial tubes become dilated, and form fusiform or cylindrical wide channels, often quite up to the surface of the lung (*bronchiectasis*).

As a result of bronchitis, the lung itself undergoes important structural changes. Acute bronchitis leads to *lobular collapse* and *broncho-pneumonia;* chronic bronchitis is followed by *vesicular emphysema,* and sometimes by *chronic interstitial pneumonia.* The last three will be spoken of separately.

Lobular collapse occurs in isolated lobules, when the bronchial tubes leading to them are blocked with mucus: since when a tube is plugged, the retained air, being stagnant in contact with the pulmonary capillaries, undergoes absorption, just as air is absorbed which has escaped into the subcutaneous cellular tissue. And, no doubt, as Fagge stated, air will get absorbed from any portion of lung in which there is no movement of air, even without obstruction of the bronchial tube which leads to it.

ACUTE BRONCHITIS.

Symptoms.—Acute bronchitis begins with some malaise, and a sensation of tightness of the chest; and cough soon occurs. In mild cases the general disturbance may be but slight, and the illness is confined to cough, expectoration of mucus or muco-pus, with very little, if any, dyspnœa. But in severe cases there is slight fever—the temperature rising to 100° or 101°, the appetite failing, the tongue furred, the bowels inactive and the urine scanty. The cough is at first hard and dry, and is often attended with pain behind the sternum and in the muscles of forced expiration from the strain put upon them. The expectoration is then but scanty, consisting of thin, frothy mucus, with, possibly, an occasional streak of blood. After a few days the cough becomes easier and looser, and the expectoration is more abundant, more opaque, and yellow or green, from the addition of increasing quantities of leucocytes. In slighter cases the expectoration is generally more in the morning, from the accumulation during sleep, and in towns this sputum is frequently black with pigment derived from the atmosphere. Dyspnœa is often considerable, with marked prolongation of expiration; the patient has to sit upright in bed (orthopnœa), and all the respiratory muscles are called into play. After a time the secretion of muco-pus becomes less, the cough is less frequent, and the symptoms gradually subside.

Physical Signs.—These are chiefly the result of the narrowing which the tubes undergo, and of the presence within them of the mucous or muco-purulent secretion. On *inspection* of the chest of one suffering from acute bronchitis, the breathing is seen to be quickened, the chest is symmetrical, and generally in a state of moderate over-distension. The accessory muscles of respiration are seen to be in strong action, and expiration is prolonged. *Percussion*, as a rule, yields a normally resonant note, but there is occasionally slight hyper-resonance from temporary over-distension of the air-vesicles; and rarely there is a little impairment of resonance at the base from accumulated secretion or from collapse. *Auscultation* shows that both inspiration and expiration are accompanied by *sibilant* or *sonorous rhonchi*, or various kinds of *râle*, or both combined (*see* p. 384). The coarser rhonchi are often felt by the hand placed upon the chest, and may be even heard by the patient himself, or those standing near him. Like the rhonchi, the râles may occur both in large and small tubes. The larger or coarser râles are heard with both expiration and inspiration, the finest râles only with inspiration. These sounds are not equally present in all cases or in all stages of the disease. In many cases rhonchi alone are present, and when both occur, the rhonchi appear first, the râles later; this is explained by the course of the changes in the bronchi already described. The sounds may be heard in severe cases, variously mixed over the whole chest, and may entirely drown the vesicular murmur. In very mild cases they may be entirely absent.

When the smallest bronchial tubes are filled with purulent secretion in the form above described as a capillary bronchitis, the position is one of extreme danger. This occurs in people of middle and advanced age,* and is often the rapid termination of a chronic bronchitis, or occurs secondarily to other morbid processes in the body. It is characterised by severe dyspnœa, great lividity of the face and extremities, and rapid exhaustion. The temperature is little, if at all, raised. The cough is at first frequent, and the expectoration is abundant, consisting of viscid glairy mucus, or muco-pus, or pus. The dyspnœa and lividity are the result of the imperfect aëration which the blood undergoes when the minute tubes are blocked with secretion; and this obstruction is shown by the retraction of the supra-clavicular, supra-sternal, and lower intercostal spaces with each inspiration. The chest is often resonant above, but the percussion note is impaired at the bases. Auscultation reveals small dull or crackling râles over the bases, back and front, almost, or entirely, masking the vesicular murmur. In later stages the patient becomes more [illegible] and drowsy; he gradually assumes the recumbent position, generally on one side. The pulse is feebler and quicker; respiratory efforts are less effectual, and the inspiratory spaces are more sucked in. Expectoration gradually diminishes, and before death the disturbance of the cerebral circulation is shown in coma, often with a slight amount of delirium.

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* The "capillary bronchitis" of children is probably, in most cases, broncho-pneumonia.

Diagnosis.—The diagnosis of bronchitis itself presents few difficulties, as it is nearly always distinguished by the sibilant and sonorous rhonchi which it causes. In the capillary bronchitis [illegible], however, rhonchi are generally more absent, and these cases are recognised by the lividity, drowsiness, absence of fever and [illegible] of the [illegible] of the two [illegible]. Rarely, [illegible] of the bronchus (p. [illegible]) may cause a stridor, which is mistaken for bronchitic râle.

The question must always be considered whether a bronchitis is primary or secondary to such other disorders as whooping-cough, measles, typhoid fever, or a part of acute miliary tuberculosis.

Prognosis.—The duration of bronchitis is from a few days to three weeks or more. In fatal cases it is from nine to twelve days, but children succumb more rapidly than young or middle-aged adults, in whom death rarely takes place from simple acute bronchitis. Capillary bronchitis is much more dangerous. Bronchitis complicating other diseases, such as cardiac lesions, the exanthemata, Bright's disease, or typhoid fever, presents essentially the same features, and, as it varies in all cases from very mild to the most severe forms, its prognosis must be considered in the same way as in the simpler forms.

Treatment.—In cases of moderate severity the patient should be placed in bed in a warm room; and much relief is often given if the air of the room be kept constantly moist by the steam issuing from a "bronchitis kettle." In the first stages of dryness and stuffiness, saline remedies, with small doses of ipecacuanha (½ to 1 grain, or 5 to 7 minims of the wine), should be given. If there is much tightness of the chest, counter-irritation by means of mustard leaf, or a linseed-meal poultice sprinkled with mustard, should be employed. The diet should be light and nutritious. In children, counter-irritants should be used with care, but a thin poultice surrounding the whole chest (jacket poultice) is of great service. In children, also, an emetic (one drachm of vinum ipecacuanhæ, repeated in fifteen minutes if necessary) is sometimes of use to get rid of accumulated bronchial secretion; and the same much more rarely in adults. As cough becomes looser, and the sputum more abundant, expectorants may be more freely used, such as ammonium carbonate (5 grains every four hours), vinum ipecacuanhæ (5 to 10 minims), or tinct. scillæ (15 minims), or a combination of two or more of these. When the cough is very irritating, sedatives may be employed—morphia in small doses ($\frac{1}{8}$ grain or $\frac{1}{6}$ grain), compound tincture of camphor ($\frac{1}{2}$ drachm), tinct. belladonnæ (10 minims), or potassium or ammonium bromide (5 grains). But they must be used with great care if there is much lividity, as they may dangerously depress the respiratory and cardiac centres under these circumstances. Cases accompanied with much spasm of the bronchial tubes may be benefited by tinct. lobeliæ (15 minims), ether (15 minims), tinct. cannabis ind. (10 minims), or potassium iodide (5 to 7 grains). Chloral in small doses (5 to 7 grains) has also been recommended. In capillary bronchitis, stimulants must be given early; and the drugs of most value are ammonium carbonate, senega (as tincture or infusion), oil of turpentine (20 minims), and lobelia. Sedatives must be given with the greatest caution or entirely avoided, for the reasons given. In all severe cases, oxygen inhalations may render valuable service.

CHRONIC BRONCHITIS.

This occurs as a sequel of the acute form, or begins in exposure or chill in those who have had a previous attack. It is common in elderly people, often appearing in successive winters, and subsiding with the approach of summer. After a time it may become continuous throughout the year.

Symptoms.—The main features of chronic bronchitis are not essentially different from those of the acute form: cough, dyspnœa, expectoration of mucus, muco-pus, or pus, with the rhonchi and râles already described. But there is an absence of fever and the constitutional disturbance which occur in acute attacks; and after long continuance, secondary results of a permanent kind are produced. In the lung itself, emphysema and dilatation of the tubes (*bronchiectasis*) take place, and these will be described later. But the effects are felt beyond the lung: the deficient aëration of the blood, which results from long-continued bronchitis, impedes the pulmonary circulation, and the right heart becomes hypertrophied; or it dilates, and thereupon the general venous system suffers, so that œdema of the lower extremities, congestion of the liver, ascites, and albuminuria occur. Under such circumstances, tricuspid regurgitation often takes place, with its characteristic murmur (*see* Diseases of the Heart). Long-continued and severe chronic bronchitis has a serious effect upon the strength of the patient. In consequence of disturbed sleep, abundant expectoration, and impaired digestion, nutrition fails, and there may be considerable emaciation. In some cases, also, in this late stage, febrile reaction of a hectic type may set in. The varieties of chronic bronchitis commonly described are the following:—

[illegible] The cough [illegible] generally worse [illegible] during the period [illegible] of the affection. The [illegible] and frothy. [illegible] may be yellow [illegible] The sputa in this [illegible] Not unfrequently, besides [illegible]

[illegible] The cough is frequent, [illegible] of the face [illegible] a small [illegible] There is much tightness of the chest, [illegible]

[illegible] expectoration is excessively abundant, [illegible] thin and transparent, or thick and ropy; it [illegible] As much as four or five pints may be expectorated in twenty-four hours, large quantities being brought up at a time with comparatively trifling effort.

5 *Putrid* or *fœtid bronchitis* is characterised by very offensive sputum, which is not associated with gangrene of the lung, but occurs especially in cases where the tubes are dilated. It will be considered under Bronchiectasis.

Physical Signs.—The auscultatory signs are similar to those of acute bronchitis; but as chronic bronchitis of long duration is commonly accompanied by emphysema, the signs of this disease may be also present (see p. 433). In cases with much accumulation of secretion the bases are dull; in such cases, also, there are abundant râles at the bases; but generally the physical signs consist of sonorous or sibilant rhonchi, more prolonged during expiration than inspiration; and of these the coarser and louder may be felt by the hand placed on the surface.

Diagnosis.—This is generally simple; the history, with the symptoms and physical signs, being sufficiently characteristic. There is occasionally a difficulty in excluding chronic phthisis, for phthisis is often accompanied by bronchitis. Here the occurrence of febrile reaction, of hæmoptysis, of rapid wasting, and the greater intensity of the physical signs on one side or at one apex, would be in favour of phthisis. It is important to recognise the cause of chronic bronchitis in cases, such as Bright's disease, where it is secondary.

Prognosis.—Though chronic bronchitis frequently shortens life, many people live to an advanced age in spite of it. It is mostly affected by the season in a marked manner, and patients are often practically well during the summer, and again get ill in the winter; but they are worse with each succeeding winter, and finally may be carried off during an exceptionally severe season, or during the cold fogs of towns, or during east and north-east winds elsewhere. On the other hand, if they can be protected from this unfavourable weather by confinement to the house, or better by residence in a warmer climate, they may keep their bronchitis within limits, and postpone the fatal termination for years. Its ill effects will, however, vary with the amount of secretion and with the rapidity with which the secondary results —emphysema, dilated tubes, and dilatation of the right heart— are developed.

Treatment.—In chronic bronchitis, as already indicated, the patient must be carefully guarded from exposure, and kept as far as possible in a uniform temperature of 60° or 65°: a moderately nutritious diet should be allowed. The medical treatment consists mainly in the use of different kinds of expectorants. Ammonium carbonate, squills, ipecacuanha, and senega may be given in ordinary cases, but particular indications have to be followed in some instances. Ipecacuanha is of use in those with hard dry cough; potassium or ammonium bromide where the cough is very irritating. In such cases, opium or morphia may have to be used, and it often gives much relief; but it must be used in small doses at all times, and withheld in cases where cyanosis is at all advanced. Where the expectoration is abundant, the balsams of Peru (20 minims suspended with 1½ drachm of honey) and tolu should be given, or ammonium chloride (5 to 20 grains); and the tendency to emaciation from the continuous drain should be met by the use of tonics at the same time. In cases where spasm of the tubes is liable to occur, lobelia, Indian hemp, sulphuric and chloric ether, and stramonium are of value, and potassium iodide has been recommended for the same purpose. Turpentine and copaiba are often beneficial in cases with free secretion; and terebene, a drug obtained by the action of sulphuric acid upon oil of turpentine, has good results in many cases. Good may be derived from inhalations of vapour of ammonium chloride, or of steam, or the use of sprays charged with succus conii, tr. benzoin co., and iodine or tar in the case of profuse expectoration. Benefit is also obtained from applications to the chest; linseed-meal poultices may be employed more or less continuously; and mustard plasters, turpentine stupes, and tincture or liniment of iodine, at longer or shorter intervals. Dry-cupping may also be of value.

If the bronchitis can be referred to any constitutional disease, this should of course, at the same time, be treated—for instance, gout by the exhibition of alkalies and colchicum. Many cases require tonics, such as quinine and cod-liver oil. It is desirable to see that the bowels are freely opened; and in cases of long standing, where the right side of the heart is dilated, the various secretions should be kept free, and the heart's action assisted by digitalis or strychnine, as under corresponding conditions in valvular disease.

The most satisfactory results are got in some cases from residence in the south of England or abroad. Torquay, Bournemouth, Penzance, Mentone, San Remo, Cannes, Arcachon, and Madeira are the places most frequented.

FŒTID BRONCHITIS.

The characteristic feature of fœtid bronchitis is the offensive odour of the sputum. It occurs most commonly as a result of bronchiectasis, when the secretions are allowed to accumulate in the dilated bronchial tubes, and so undergo decomposition. But it may arise under other conditions—for instance, in acute bronchitis—and the cause is probably in most cases the entrance of bacteria by means of inspired air.

Symptoms.—The sputum is abundant and rather thin, and in the sputum-vessel it separates into three layers, of which the uppermost is muco-purulent and frothy, the middle a thin seromucous fluid, and the lowest a thick layer of pus containing the bodies known as Dittrich's or Traube's plugs. These are whitish-grey or dirty greyish-yellow, varying in size from a millet-seed to a bean. Under the microscope they are seen to consist of pus-corpuscles, detritus, bacteria, bundles of fine acicular crystals of palmitic and stearic acids, and twisted threads of leptothrix; but neither lung tissue nor specific organisms occur in the sputum. Chemically, it is found to contain acetic, butyric, and valerianic acids, leucin, tyrosin, sulphuretted hydrogen, and methylamine. The odour is very offensive, putrid, and at the same time somewhat sweet; not only the sputum but the breath of the patient is charged with it, so that none can stay near him, and it pervades the atmosphere of the room. The onset of the putrid change may be accompanied by febrile reaction, prostration, and loss of appetite; and lobular pneumonia and gangrene of the lung may occur as secondary results. But the condition may become chronic, with only occasional attacks of pyrexia; loss of appetite, nausea, and indigestion may then occur, but otherwise no serious impairment of health. Painful swellings of the joints have also been recorded. Some patients recover completely.

Pathology.—In fatal cases one generally finds dilatation of the bronchial tubes of old date, and the mucous membrane is intensely injected or ulcerated. The lung may present lobular pneumonia or gangrene.

Diagnosis.—The distinction from gangrene is difficult, and all the more so as the two may coexist. The physical signs of fœtid bronchitis are mostly those of bronchitis with dilated tubes—namely, fine or medium râles; while in gangrene one gets more often signs of consolidation or excavation. The presence of lung-tissue in the sputum would be in favour of gangrene, but it is not so constantly found in that condition as one would expect.

Treatment.—This must be of a supporting and stimulating kind, while locally we endeavour to lessen the decomposition and diminish the fœtor by antiseptic inhalations. Carbolic acid, creosote, turpentine, thymol, eucalyptol, and tincture of iodine, may be used, either inhaled from the surface of hot water, or administered by means of a respirator (*see* Treatment of Phthisis). Counter irritants and blisters may also be of value.

Plastic Bronchitis.

(*Fibrinous Bronchitis. Croupous Bronchitis.*)

This affection is characterised by the expectoration of more or less perfect casts of the bronchial tubes. The sputum is generally in the form of a rounded mass, covered with mucus or blood, and, when frayed out in water, one sees a perfect branching cast of a portion of the bronchial tube system. The cast is not generally thicker than a goose-quill, and varies from one and a half to two and a half inches in length, and only rarely reaches four or five, or even seven inches. It has a grey or whitish-yellow colour, and consists of concentric laminæ, which do not usually fill up the lumen of the tube, so that the casts are not solid, except those from the very smallest tubes. Under the microscope the cast has a fibrillated structure, with numerous imbedded leucocytes, hæmatoidin crystals, Curschmann's spirals, or Charcot-Leyden crystals (*see* p. 428).

Ætiology.—The disease is extremely rare; it occurs in males more frequently than in females, and mostly begins between the ages of ten and forty; it has also been noticed to occur in different members of the same family.

The **Symptoms** preceding the expectoration of the casts may be very slight; indeed, the patient may appear to be in ordinary health; or there may be indications of bronchial catarrh, or a pyrexial condition, with rigors suggestive of pneumonia. Then the patient is seized with violent attacks of coughing, often suffocative in character, with more or less pain or oppression at the chest, and attended at first with no sputum, unless perhaps a little mucus. After a time—it may be a few hours, or as long as two or three days—a bronchial cast is brought up. Relief is generally at once afforded: the cough subsides or disappears. But it commonly recurs in a few hours, and casts may continue to be expectorated, at intervals of a day or so, for several days, when the patient gradually gets quite well. Hæmoptysis occurs in some cases, usually after the expulsion of the cast. The physical signs are attributable to the obstruction of the tube or tubes. The vesicular murmur is commonly deficient; and there may be either slight increase of resonance over the area of lung corresponding to the obstructed tube, or, on the other hand, dulness from collapse of the same portion. The movements of one side of the chest may be impeded if tubes are extensively blocked; and râles are sometimes heard as the casts are becoming loosened.

Prognosis.—The disease is rarely fatal, except from complications: but it recurs at irregular intervals over a period of several years.

The **Treatment** hitherto tried has been unsatisfactory. Iodide of potassium in full doses is credited with some success; mercurials, tartar emetic, alkalies and their carbonates, have been at different times used. Inhalations of steam and tar vapour, and sprays of lime-water and of alkaline carbonates, are also recommended; and an emetic is said sometimes to be of value.

BRONCHIECTASIS.

Dilatations of the bronchi may occur in connection with many diseases of the lungs such as bronchitis, emphysema, bronchial or tracheal obstruction, chronic pneumonia, phthisis and pleurisy: they may be so slight as to be masked by the symptoms of the disease which has preceded them, or so large as to form the prominent feature of the case. Rarely the condition is congenital.

The effect of a continued bronchitis is to soften the tissues of the bronchial wall, and lead to more or less atrophy of the muscular fibre and of the cartilages. The bronchial wall is then likely to yield to the pressure of the air in coughing efforts and other strains, or to the pressure of the secretion as long as it is retained. A similar result follows the obstruction of a main bronchus, such as may result from aneurysm, or stenosis by syphilitic cicatrix, or cancer. In ordinary cases the change affects the medium and smaller tubes; they are dilated into somewhat irregular cylindrical tubes (*cylindrical bronchiectasis*), and can be traced with the greatest ease nearly to the surface of the lung; they are more common on the lower lobes than in the upper.

Though the process is usually a chronic one, an acute bronchiectasis of numerous smaller tubes throughout the lung is occasionally seen after an acute catarrhal bronchitis.

In other cases the dilated tubes are oval or globular (*saccular bronchiectasis*). In this variety a number of smooth-walled cavities, from the size of peas to marbles, or larger still, are found in the lungs; their walls are thin, and present generally no trace of the muscular tissue or cartilage of the healthy bronchi; a small bronchus may often be found opening into the cavity. Sometimes there are bands running along the walls; sometimes the surface is ulcerated.

This kind of cavity is frequently associated with the extensive fibroid changes found in chronic pleurisy and fibroid phthisis, and the contraction of the fibrous tissue tends to the enlargement of the cavities. They are situated much more commonly at the base and the middle of the lungs than at the apex.

Symptoms.—In cases of moderate cylindrical dilatation associated with bronchitis or emphysema, the symptoms will be lost in those of the primary disease. But in larger dilatations and in the saccular variety the secretion from the cavities and the fibrosis of extensive portions of the lung are productive of definite symptoms, namely dyspnœa, cough, and expectoration.

The patient need not be emaciated, is generally free from fever, and may be inconvenienced by little besides the cough and sputum; but he is somewhat cyanotic, the fingers are clubbed at the ends, and in course of time the effect of the pulmonary lesions upon the right heart will cause œdema of the feet, enlargement of the liver and albuminuria.

The most characteristic feature is the way in which the sputa are expectorated. The secretion is liable to collect for some time—it may be two or three hours—in the dilated tubes, without exciting cough; then either from its quantity, or because the patient moves about, turns over or sits up in bed, the secretion flows over into an adjacent healthier tube, coughing is excited, and some ounces of muco-purulent secretion are all at once expectorated. In many cases of bronchiectasis the sputum becomes fœtid (*see* p. 422).

Physical Signs.—With cylindrical tubes and not much fibrosis there are various small and medium-sized râles, audible especially at the bases. If there is consolidation of the intervening lung, from collapse or chronic pneumonic processes, tubular breathing will be heard. In the extremer cases with much fibrosis, the conditions of chronic pneumonia are produced. The side most affected is retracted, especially in its lower and middle parts, there are impairment of resonance, coarse bronchial or cavernous breathing, and creaking râles, distributed more or less extensively, but always as much or more at the mid-level and base, than at the apex. The opposite lung may be partly emphysematous to compensate. With extensive contraction of the lung, the heart will be drawn horizontally to the affected side, and the right ventricle will become dilated with results just indicated.

Diagnosis.—In the advanced cases there is a resemblance to phthisis. The chief points of distinction are that in bronchiectasis the physical signs are not confined to, nor most marked at the apex, the patient is not febrile, nor ill and emaciated in proportion to the extent of the local mischief, and there are no tubercle-bacilli in the sputum.

Prognosis.—As compared with phthisis it is good; patients often live for years with but slow advance in the local conditions.

Treatment.—The patient requires support by means of tonics, such as iron, quinine, and cod-liver oil, and should be placed under the best hygienic and climatic conditions on the same principles as in the treatment of phthisis. Locally the object should be to assist the evacuation and the disinfection of the secretions. Inhalations of antiseptics (*see* Bronchitis) are of value; but other more thorough means have recently been tried, as for instance, Poore's administration of garlic internally, by means of capsules containing 30 grains of chipped garlic; the intra-tracheal injection twice daily of 1 drachm of a solution of 10 parts menthol, 2 parts guaiacol, in 88 parts olive oil; the daily inhalation for 15 to 60 minutes of the vapour of creosote in a closed chamber (Chaplin).

ASTHMA.

In asthma the patient suffers from sudden attacks of dyspnœa, which subside after a time, and recur at irregular intervals. The dyspnœa results from obstruction of the smaller bronchial tubes, probably due to spasm of the muscular fibres in their walls.

Ætiology.—One must consider, first, the causes of the disease itself—that is, of the tendency to suffer from the paroxysms of dyspnœa; and secondly, the various circumstances which may bring on an attack in those disposed to them. The disease is often transmitted hereditarily, but it may not in such cases show itself till an advanced age. In children, who contribute a large proportion of asthmatic cases, it may follow measles, whooping-cough, or bronchitis. Adenoid growths in the nose and naso-pharynx sometimes cause asthma. A neurotic origin is often noticed. Salter observed that it might alternate with epileptic attacks, whilst it is sometimes associated with neuralgia, angina, or other neuroses in the same person. Gout rather predisposes to asthma, and malaria and syphilis have been held responsible for some cases; moreover, it sometimes occurs on the subsidence of skin eruptions, such as eczema and lichen. The disease begins at all ages; it is twice as frequent in males as in females.

Amongst the exciting causes of an attack are a number of impressions upon the peripheral nerves, or more central disturbances of the nervous system; and the greatest variety exists as to the way in which, in different persons, the attack is brought on. Particular climates or atmospheres, cold air, the close atmosphere of a badly ventilated room; particles of dust or fluff; smoke; the odour of hay, of certain flowers, or of ipecacuanha; or the emanations from some animals—namely, cats, rabbits, dogs, horses, &c.—may at once produce it. Diet has an important influence: any overloading of the stomach may set up an attack in some persons, but a late meal is especially likely to do it; or certain articles of diet have to be carefully avoided. Constipation and uterine troubles occasionally act as irritants. Emotion, anger, and fright are instances of cerebral disturbances causing asthma.

Symptoms.—Sometimes the attack is preceded by *premonitory* indications, such as a general sense of discomfort, drowsiness, gaping, itching under the chin, sneezing and coryza, or the passage of much pale limpid urine. But it is often quite sudden, commencing in the early morning between two and four o'clock, though the patient may have gone to bed apparently quite well. He wakes up with a sense of dyspnœa, so that he has to sit up in bed, or gets out and opens the window to let in more air. The breathing is soon so difficult that he has to call in the aid of all the accessory muscles of respiration; he grasps with his hands the sides of his bed, the arms of a chair, the mantelpiece, or the edge of a table, to give a fixed support for the muscles which pass from the upper extremities to the chest. The chest, however, is nearly fixed in a condition of inspiration, and there is very slight movement. The respiration is often quite slow, but occasionally rapid; the most noticeable feature is the extraordinary length of expiration, which is accompanied with a loud wheezing, audible at a distance. The chest is somewhat over-resonant; the inspiratory murmur is scarcely audible, or accompanied with a little sibilant rhonchus, while with expiration is heard the loud rhonchus just mentioned. With this the patient's distress is very great; the face gets cyanosed, the eyes are prominent, the conjunctivæ suffused, and the whole attention of the patient is absorbed in the attempt to get a proper interchange of the air in his chest. Usually there is no pyrexia. After a time—it may be two or three hours—he begins to cough, and expectorates some thin, transparent mucus, which may be mixed with a little blood; then the breathing becomes easier, the cyanosis is less, gradually the whole trouble subsides, and the patient falls off to sleep.

The sputum often contains, besides cylindrical or ciliated epithelium, two peculiar constituents—namely, Curschmann's spirals, and octahedral crystals. The former are yellowish-green or grey particles, made up of threads of mucus; under the microscope they are seen to be spirally twisted fine or coarse fibres, and there is often in the middle one fine transparent fibre. The octahedra may be present in the spirals; they are known as Charcot-Leyden crystals, and consist of phosphate of spermin. Similar crystals are found in the blood in leuchæmia, and sometimes in the fæces in dysentery, typhoid fever, and other conditions.

Each attack of asthma may last from two or three hours to as many days; their recurrence, at longer or shorter intervals, is a good deal determined by the exciting causes—that is, a careful patient, who knows how to avoid what will bring on his attacks, may escape for long periods. As to the duration of the illness, that also is very variable. Many of those who have it in childhood recover in adult age; but those who acquire it in middle age never recover. The attacks themselves are rarely fatal, and the occasional occurrence of not very severe attacks is not prejudicial to health; but frequent paroxysms induce emphysema of the lungs, and ultimately attendant bronchitis, so that there is constantly more or less lividity, with the round shoulders, barrel-shaped chest, and laboured respiration which are observed in the midst of the paroxysms themselves. Life is thereby shortened, and the tendency to suffer from the severer forms of bronchitis is increased.

Pathology.—The attacks are clearly attributable to some obstruction of the minute bronchial tubes, and the prevailing view is that this obstruction is due to spasmodic contraction of the bronchial muscular fibre; hence the name "spasmodic asthma," which contrasts with the popular use of the term asthma for every kind of dyspnœa, especially chronic bronchitis and emphysema. But the disease has also been attributed to vascular or erythematous swelling of the bronchial mucous membrane. The sudden development of the symptoms and the comparatively rapid subsidence in many cases are in favour of its spasmodic origin.

Treatment.—Climate is one of the first things to be considered. A large number of patients can live in London and large cities free from paroxysms, who have them at once if they attempt to live in the country. Conversely, some can only live in the country, and have asthmatic attacks in town. In the same way sea-air may excite attacks in some, and cure others. The facts with regard to any patient can only be ascertained by experiment.

Moderation and care in diet are the next points to consider. Food should be light and easily digestible; a heavy supper should not be taken; and particular foods should be excluded from time to time, such as cheese, pastry, pork, beer, to see if there is any one offender in this respect. If the trouble cannot be met in these ways, and the naso-pharynx presents no lesions for surgical treatment, potassium iodide should be given in doses of from 5 to 10 grains three times a day, continuously and irrespective of the attacks, for some weeks or months; and arsenic is similarly useful. A number of remedies have been used in the attacks, and many of them are decidedly effectual in lessening the severity of the dyspnœa, and shortening the paroxysm. The most useful seem to be those which are inhaled, and so possibly act directly upon the bronchial tubes. The vapours of chloroform, ether, nitrite of amyl, iodide of ethyl, and turpentine may thus be used; but more lasting results are often obtained by the fumes from burning a paper saturated with nitre-solution and dried, or by smoking cigarettes made of chopped stramonium-leaves, or by the use of other preparations containing stramonium. Some similar drugs may be given internally, such as nitro-glycerin, and nitrite of sodium—which also paralyse organic muscular fibre—and chloral, morphia, potassium bromide, antipyrin, extract or tincture of stramonium, belladonna, or lobelia. Two or more remedies may be combined. Local applications may give some relief, such as mustard plasters or turpentine stupes, and, according to some, the application of iodine tincture on the side of the neck, over the course of the pneumogastric nerves. In chronic cases which resist treatment, general tonics, like quinine, or cod-liver oil may be of value, and the patient should be careful not to expose himself unduly, in view of the secondary changes in the lung which supervene.

OBSTRUCTION OF THE LARGE BRONCHI.

In their relation to the various causes of compression, the two main bronchi closely resemble the trachea, and much that has been said under the head of tracheal obstruction might be repeated here. Aneurysms and mediastinal growths are the chief causes of compression; syphilis leads to stricture; and foreign bodies become impacted. One or both bronchi, and perhaps at the same time the lower end of the trachea, may be compressed by growth or aneurysm. The special liability of the left bronchus to compression by an aneurysm of the arch of the aorta, under which it passes, is of importance. Foreign bodies more frequently fall into the right bronchus, because the dividing ridge between the two bronchi is somewhat to the left of the middle line, and hence objects falling down the centre of the trachea are directed into its right branch. They may be driven into the trachea during coughing, and fall back into the same or the opposite bronchus. If the object is impacted it proves a permanent obstruction, and may cause hæmoptysis or ulceration and sloughing of the mucous membrane.

Symptoms.—These vary with the degree of obstruction; and since the opposite tube is often free, and thus only half the respiratory area is interfered with, the bronchus is often much more completely obstructed than the trachea can be. There is commonly dyspnœa, and occasionally stridor. In the absence of stridor, the vesicular murmur over the corresponding lung is found to be very deficient or absent; and tactile vibration is diminished, though the percussion note over the chest remains normal. In extreme cases all the air may become absorbed from the lung, which becomes completely collapsed, while the chest shrinks, with percussion dulness and entire loss of breath and voice-sounds and vocal fremitus. Another result of stenosis of a bronchus is a condition of more or less extensive bronchiectasis, with secondary fibroid thickening of the lung or pleura. Pus forms abundantly in the cavities, and may be expectorated in large quantities at a time. The physical signs may be those of a pulmonary cavity—hollow bronchial breathing, bronchophony and pectoriloquy. Sometimes the lung becomes pneumonic or gangrenous. Coupland has recorded a case of obstruction in which the dilated tubes communicated through one of the intercostal spaces with the subcutaneous tissue on the front of the chest, and an abscess was opened, from which a quantity of offensive pus was discharged. Foreign bodies do not always confine themselves to producing mechanical obstruction, but they have occasionally set up diffuse suppurative pneumonia, or have worked their way to the surface of the lung, perforated the pleura, and caused pleurisy or pneumothorax.

Diagnosis.—The combination of good resonance with nearly complete absence of respiratory sounds on one side of the chest is very characteristic of obstruction of the corresponding bronchus. When the obstruction is accompanied by stridor, it may be mistaken for bronchitis. Stridor from the above cause is persistent and uniform in character, arising from a single point of obstruction, whereas the rhonchi of bronchitis vary constantly in loudness, pitch, and position. Bronchitis further is more often bilateral. If the lung has shrunken from the stenosis, the physical signs resemble those due to a partially absorbed pleuritic effusion. Here the history will help; but the use of the exploring syringe may be necessary to decide.

The **Prognosis** and **Treatment** are similar to those of obstructed trachea (p. 414); tracheotomy is of course useless except for the removal of foreign bodies.

DISEASES OF THE LUNGS.

EMPHYSEMA.

The term emphysema (from ἐν, in, and φῦσα, wind) is rightly used to denote the extravasation of air into the subcutaneous or other tissues of the body (*surgical emphysema*), and into the interlobular or interstitial tissue of the lungs (*interstitial emphysema*). It is much less applicable to the disease of the lung now under consideration, for which, however, in medical parlance it is usually reserved. The alveoli of the lung naturally contain air; in this disease they are abnormally distended, and may be said to contain too much. So far the name emphysema (*vesicular emphysema*) may be justifiable; but a more correct name would be that of *alveolar ectasis*, which has been suggested.

Anatomy.—The essential change in emphysema of the lungs is a loss of elasticity, from weakening, and subsequent atrophy and destruction, of the elastic tissue contained in the alveolar septa. In consequence of this the walls of the air-vesicles yield to the pressure of the contained air, and become distended. This brings them into closer contact with neighbouring alveoli which are also dilating; and between the two the alveolar septum becomes atrophied. Soon a perforation is established through the septum between the two alveoli, then the whole septum is destroyed, and the two alveoli become one. In this process not only the elastic tissue, but also the whole network of pulmonary capillaries contained in the septum, disappears. If this is repeated extensively throughout the lungs, first, all the air-spaces are much enlarged, and in many places great blebs of lung-tissue simply containing air are formed; secondly, the elasticity of the lung necessary for expiration is reduced much below the normal; thirdly, the vascular area available for aërating the blood is greatly diminished; and fourthly, in most cases the lungs themselves are considerably enlarged. A lung affected with emphysema does not collapse when the chest is opened at the *post-mortem* examination, but even bulges out through the aperture. It is soft, and inelastic, and yields to the pressure of the finger ("pitting"). In different parts of it, especially along the inner and lower edges, may be seen large blebs the size of peas or nuts; and the lung is unusually pale, and bloodless, and of a mottled grey colour. On section the larger blebs collapse, and the whole organ is much drier than usual, unless in some parts, such as the bases, which may have been the seat of a complicating bronchitis or œdema.

A variety (small-lunged emphysema) occurs in old people, as a senile atrophic change; the lung is not enlarged, and blebs are not numerous. The septa have atrophied so that alveoli have joined together, and the lung is shrunken, inelastic, drier, and paler, and presents a less perfectly spongy structure than normal.

Ætiology and Method of Production.—No doubt many cases of emphysema result from bronchitis, and some from whooping-cough or asthma, but this will not account for all, as it is certainly common to find the indications of a slight emphysema in those who have never had any such illness. In all cases it is the failure or wearing out of the elastic tissue that is the essential lesion.

This may develop with advancing age as a senile change; it may be more directly induced by a number of laborious occupations which entail prolonged strain upon the lungs, the chest being held full of air for a long time, either to serve as a *point d'appui* for the use of the arms, or to supply air in a regulated way, as in playing upon wind instruments, glass-blowing, &c.; but it is sometimes found in young people whose circumstances do not seem to be in any way exceptional.

The mechanism in the case of glass-blowers and others, is probably that the lungs are kept expanded during the regulated effort, or that the force of expiration is opposed by the obstruction in the work, and so the elastic tissue is kept unduly on the stretch. Prolonged coughing in bronchitis and whooping-cough has the same effect; and in the former the secretions constitute an obstruction to the expiration, from which the elastic tissue necessarily suffers strain. The greater development of emphysema in certain parts of the lungs, especially the anterior margins and lower edges, and in the neighbourhood of old cicatrices at the apex, may be accounted for on Jenner's view—that when air is retained in the chest under great pressure, as when playing a wind instrument, or making any great muscular effort, it is the parts of the lung which are least supported by the surrounding structures which will be most subject to the air-pressure from within. These are precisely the anterior and lower edges in healthy lungs. When a portion of lung shrinks from old disease, the support of the adjacent lobules is, of course, withdrawn; and the local emphysema which thus arises is called *compensatory*.

Results of Emphysema.—These are of two kinds. In consequence of the *loss of elasticity*, expiration, which is largely effected by the spontaneous collapse of the lungs, after inspiration, becomes more difficult; the lungs tend to increase in size; the chest enlarges in width and depth, assuming permanently the shape and position which are characteristic of full inspiration; the mobility of the chest is much diminished, since it ranges only between different degrees of inspiration, instead of between full inspiration and full expiration; the interchange of gases is less complete; and every attack of bronchitis is aggravated from the impairment of coughing power which follows on the above defects.

The other important factor is the *loss of capillary area*, and hence of aërating surface. From this results an obstruction to the pulmonary circulation, of a kind similar to that which is produced by disease of the left side of the heart. The tension in the pulmonary artery and right ventricle is increased, the right ventricle hypertrophies or dilates, or both, and the venous system becomes engorged, producing in course of time congestion and enlargement of the liver, œdema of the feet, legs, and trunk, and albuminuria.

Symptoms and Physical Signs.—The symptoms of emphysema are at first only shortness of breath; the cough and expectoration which are commonly present result from a co-existing bronchitis. The dyspnœa is especially seen on exertion in early stages, when the breathing is quickened and the patient readily pants; later on, it may be always present, producing orthopnœa at night. In its worst forms the extraordinary muscles of respiration are in constant use; the clavicles are lifted, and the sterno-mastoids and scaleni stand out at each inspiration, striving to increase the tidal air; expiration is prolonged, laboured, and aided to their utmost by the muscles of the abdomen. The *physical signs* are characteristic. The chest is broad, deep antero-posteriorly, but short; it is often called barrel-shaped, from its enlargement, and from the increase of the antero-posterior diameter giving it rather a circular than a transversely oval shape. The shoulders are raised; the upper ribs are closer together, and the lower ribs wider apart than normal; and the epigastric angle is very obtuse, measuring 105° or more. The elevation of the ribs alters the relative positions of the nipple and the heart's impulse; the nipple is often found on the fifth rib, and the heart's impulse in the sixth space. But this last may be partly displaced by the enlarged lung. Percussion gives excessive resonance over the parts of the chest that are normally resonant, and an extension of the resonance over areas that are normally dull. Thus the hepatic and cardiac dulnesses are encroached upon, the right lung being resonant down to the sixth space or seventh rib, and the heart-dulness being limited to the fifth cartilage and space below, or even disappearing altogether. Posteriorly, the resonance extends to its fullest limits downwards. On auscultation the inspiratory murmur is very much diminished or scarcely audible, and the expiratory murmur is much prolonged.

The enlargement of the lungs also affects the signs connected with other organs. The impulse of the heart is scarcely perceptible in the normal position, but epigastric pulsation is often present. The cardiac sounds are much less loud, from the lung overlying the heart. Any enlargement of the heart from dilatation or hypertrophy of the right ventricle may thus be concealed. The systolic murmur of tricuspid regurgitation is occasionally present. The liver and spleen may be slightly displaced downwards.

In small-lunged emphysema the chest is barrel-shaped, but not enlarged; the lungs do not cover the heart; and the heart is not hypertrophied, but atrophied. The percussion note is hyper-resonant, and the inspiratory murmur is feeble, but the expiration is not prolonged.

In both forms the signs of bronchitis frequently co-exist—namely, sibilant and sonorous rhonchi; in extreme cases there are râles at the bases of the lungs, and the intercostal spaces are sucked in with inspiration.

Complications.—Chronic bronchitis is most frequently present, with or without bronchiectasis; and in late stages of the more common form, dilatation of the right ventricle, with anasarca and albuminuria. Bright's disease, with its cardio-vascular changes, may complicate it in old patients.

Diagnosis.—Its recognition depends upon the altered quality of resonance, and especially by the extension of resonance over the præcordial area, and downwards over the liver. In the small-lunged variety the altered quality of resonance and the dyspnœa are the chief features.

Prognosis.—Actual recovery does not occur; only relief of symptoms. The duration of life depends upon the extent of the change, and the liability to bronchitis, or to cardiac dilatation. In most cases, the final result does not come under several years.

Treatment.—This must be directed to improving the general health of the patient, to avoiding all risk of bronchitic complications, and to relieving these when they occur. Thus the patient should have nutritious and digestible diet, should be well clothed, live in warm, well-ventilated rooms, and avoid east winds and the night air. Tonics, such as cod-liver oil, iron, strychnia, and quinine, are of some value, since the disease partakes of the nature of degeneration. Attempts have been made to compensate for the loss of elastic tissue by different methods; thus Gerhardt advises assisting expiration by mechanical compression of the thorax; this is done by another person with the hands upon the lower part of the thorax for five or ten minutes every day. A patient of Strümpell's managed it himself by means of two boards, one on either side of the chest, which he brought together at each expiration. The compressed air bath, in which the patient breathes air at an additional pressure of two-thirds of an atmosphere, has been found useful at the Brompton Hospital.

For the accompanying bronchitis, expectorants such as ammonium carbonate in doses of 5 to 7 grains, vinum ipecacuanhæ, and the infusion or tincture of senega, should be given. Mustard plasters or linseed meal poultices will afford some local relief. If the heart is failing, digitalis, strychnine, or other heart tonic must be used; and when anasarca is threatened, purgatives such as pulv. jalapæ comp., and diuretics, such as squill, acetate of potassium, spirits of nitrous ether, and citrate of caffein, should be given to relieve the overloaded venous circulation.

COLLAPSE OF THE LUNGS.

(*Atelectasis Pulmonum.*)

A distinction is often made between lungs that have never completely expanded (atelectasis) and those that have, after expansion, partly returned to the fœtal state (collapse). The former condition is congenital, and is seen in very weakly children, whose respiratory movements are insufficient to draw in air to the necessary extent. The latter results from anything, which by obstructing the entrance of air, or by compressing the lung from without, prevents its complete expansion. *Obstruction* may arise from chronic enlargement of the tonsils, adenoid growths in the naso-pharynx, much more often from the viscid, mucous, or purulent secretion of bronchitis, especially in children, and as a part of broncho-pneumonia, and from constriction of the bronchus by cancer or by aneurysm in older people. The causes of *compression* are numerous; in the chest itself it is most frequently due to pleural effusion, but also to enlargement of the heart, pericardial effusion, mediastinal tumours, aneurysms of the aorta, and angular curvature of the spine (kyphoscoliosis); in the abdomen, to the pressure of tumours growing from the upper surface of the liver, especially hydatids, abscess and cancer, of subdiaphragmatic abscesses, hydatid of the spleen, ascitic fluid, and ovarian tumours. The mechanism of compression is obvious; in the case of obstruction it has been pointed out that when the to-and-fro movement of the air in the lungs is prevented, the air imprisoned within the air-vesicles of the lung becomes absorbed, and the vesicles collapse.

Morbid Appearances.—Lung in a state of collapse or atelectasis has a violet or dark purple-grey colour, is tough, airless, and dry on section. Isolated patches are seen to be slightly depressed below the general surface. Unless subsequently the seat of inflammation, they may be again expanded by forcible inflation with air.

Symptoms.—In congenital atelectasis the child is weakly, more or less livid, with rapid shallow breathing and feeble cry. The lower part of the chest is drawn in with each inspiration, and the intercostal spaces are depressed. Examination may elicit a little loss of resonance at the bases, and occasionally some râle, but feebleness of breath-sounds is the chief physical sign. The collapse of bronchitis is rarely extensive enough to reveal itself by auscultation, its distribution being lobular and scattered. In the frequent cases of compression by fluid, &c., the physical signs of collapse form an essential part of those of the primary disease; they simply represent the negation of those signs which are due to the presence of healthy lung—that is, they consist in the absence or diminution of resonance, of breath-sounds, of voice-sounds, and of tactile vibration.

There are, however, two earlier or incomplete stages which must be recognised. One is the state of partial collapse, which may result from temporary disuse of a portion of lung, as, for instance, during a slight pleurisy, or from lying upon the back for a time. When the affected area of lung is auscultated. the breath-sound is very feeble; if the patient breathes deeply, there is a louder vesicular murmur, and at the end of it a fine dry rustling râle (crepitation), which is due to the fresh expansion of hitherto collapsed air-vesicles.

The other stage is that in which, from partial pressure of fluid, the lung tissue is compressed, but the bronchi remain patent; the physical signs then may be bronchial breathing and bronchophony, deceptively like those of pneumonia. This is not infrequent as a result of pleurisy, but it may also occur from pressure backwards of a large heart or distended pericardium.

Treatment.—In most cases of collapse, the primary cause must be discovered and treated. In congenital atelectasis, the treatment must be supporting. The child should be kept warm in a well-ventilated apartment, the chest may be gently stimulated by friction, and the food should be looked to. In older children, bronchitis, rickets, or congenital syphilis must be met by appropriate treatment.

ŒDEMA OF THE LUNGS.

This consists of the exudation of serous fluid into the interstices of the lung. and into the air-vesicles and smallest bronchi.

Ætiology.—It results from disturbances of the circulation, and from general blood diseases; and its most frequent causes are valvular disease of the heart, and acute and chronic Bright's disease. It is not infrequent in elderly people with failing hearts, as a more or less permanent condition; it forms part of the condition known as hypostatic congestion in prolonged febrile illnesses, such as enteric fever; and an inflammatory œdema generally accompanies acute pneumonic processes. Local œdema may result from the pressure of tumours or of aneurysm on the pulmonary vessels: and finally, in death from diseases involving the lung, like pneumonia or pleuritic effusion, œdema of the formerly healthy lung contributes to the fatal termination.

Symptoms.—The symptoms which are due to the œdema, in addition to those of the primary lesion, are dyspnœa, orthopnœa, more or less cyanosis in extreme cases, cough, and expectoration of abundant frothy serum, or sero-sanguineous fluid. The chest is resonant, or at most shows some slight impairment of the note at the bases behind; here the breath-sounds are deficient, and there are heard only abundant fine and medium râles. In the final œdema of pneumonia, the râles are audible over the whole of the hitherto healthy lung.

Morbid Anatomy.—A lung affected with œdema is bulky, heavy, and exudes when incised an immense quantity of serous, slightly blood-stained fluid.

Treatment.—This is chiefly to be directed to the primary cause. Heart disease requires prompt treatment by the usual cardiac tonics, diuretics, and purgatives. In Bright's disease, diaphoretics and purgatives, or the vapour bath should be employed. In elderly people with failing hearts, cardiac and general tonics are desirable. Ammonium carbonate is valuable in œdema of the lung, either by acting as a direct expectorant, or by stimulating the contractions of the left ventricle, and thus facilitating the circulation of blood through the lung.

PNEUMONIA.

Inflammation of the substance of the lung, as opposed to the bronchial tubes, is called pneumonia. As an *acute* disease it leads to consolidation by exudation into the air-vesicles of inflammatory products, which may be afterwards absorbed. In a *chronic* form it causes a dense fibrous transformation of the interstitial tissue, which is permanent. Of acute pneumonia, two typical forms can be distinguished from one another by the following features:—*Croupous* pneumonia occurs especially in adults, affects large portions of the lung at the same time, and is hence called *lobar*, and has all the characteristics of a specific infectious disease, with a limited duration, termination by crisis, and sometimes epidemic prevalence. *Catarrhal* pneumonia or *broncho-pneumonia* affects chiefly infants and young children, invades several small areas of the lung, having a *lobular* distribution, and is much less definite in its course and modes of onset and termination. The two forms differ also in their histology and bacteriology. These distinctions are, however, by no means absolute, and some of the difficulties of recognition and distinction will be pointed out after the two have been separately described.

Chronic or *interstitial pneumonia* constantly forms a part of chronic phthisis and bronchiectasis, and only in a small number of cases stands alone as the primary lesion.

CROUPOUS PNEUMONIA.

(*Acute Lobar Pneumonia.*)

Ætiology.—The disease occurs in both sexes and at all ages, but statistics have shown that it is twice as common in males as it is in females, the difference between the two sexes being least marked in the very young, and in old people. It is more frequent in adults up to middle age than at other periods of life, and this contrasts with the greater frequency of broncho-pneumonia amongst quite young children and old people. Pneumonia occurs much more often in the winter and spring than in the summer and autumn; when the temperature is undergoing sudden changes, when the winds are east or north-east, or when the weather is wet or cold. Habits and occupations which involve exposure predispose to pneumonia, and it is probable that persons of weak health, or suffering from mental depression, or those who do not have sufficient nourishment, are more liable to it than the robust and strong. Intemperate habits dispose to it, and greatly increase its mortality. An attack does not exempt from another; indeed, pneumonia is said to have occurred as many as fifteen or twenty times in the same patient, but more than two attacks are not very common.

Amongst direct exciting causes, cold or chill is much less often the cause than has hitherto been believed. The influence of insanitary conditions has still to be determined; and also the question whether pneumonia may arise from contagion, and the nature of the epidemics of pneumonia. A large number of instances have been recorded of late years in which pneumonia has spread rapidly through villages, large buildings, or households, precisely like an epidemic fever, and cases of apparent infection have also been observed.

Croupous pneumonia also occurs as a complication or sequela of some other diseases, and especially of mitral disease, acute nephritis, some infectious diseases, and diabetes. Pneumonia is no doubt a specific infectious disease, with the primary seat of infection in the lung. This is shown by the mode of onset and termination, the occurrence of epidemics, and the constant presence of a specific micro-organism in the lungs and sputum. This is the diplococcus pneumoniæ of Fränkel, diplococcus lanceolatus, or pneumococcus; but other organisms are also found in smaller numbers—namely, Friedländer's bacillus, and streptococci and staphylococci. The pneumococcus is not peculiar to pneumonia; it is found in association with inflammation of other structures, such as the pleura, meninges, and endocardium.

Morbid Anatomy.—In pneumonia, the part of the lung affected is converted from a spongy structure into a solid mass. In the earliest, or first stage of *congestion* or *engorgement*, the lung is heavy, reddish-brown in colour, exudes a frothy, reddish serum on pressure, and breaks down more readily than in health. The capillaries are dilated and tortuous from distension with blood, and minute hæmorrhages may be present. In the second stage—called the stage of *red hepatisation*, from the resemblance which the consolidated lung bears to the liver—the organ is of a dull red colour, finely granular on section, completely airless, solid, sinking in water, but breaking down readily under the pressure of the finger. The contents of the alveoli, which may be detached in fine granular masses, are seen to consist of coagulated fibrin, containing red blood-corpuscles and a few leucocytes. The third stage, *grey hepatisation*, is also characterised by its solidity, but the colour is greyish-yellow or simply grey, and the surface is less granular than that of the red stage. Microscopically, it differs from the latter in that the air-cells and alveolar walls are crowded with leucocytes, while fibrinous exudation and red corpuscles are in very small quantity. The change of colour is attributable to the leucocytes in the alveolar walls, and to the decolorisation of formerly extravasated red corpuscles, but mainly to the blood in the vessels of the alveolar walls being prevented circulating by the pressure of the infiltration. Rindfleisch states that this bloodlessness only occurs after death, and hence that grey hepatisation is chiefly a *post-mortem* condition. A fourth stage, that of *purulent infiltration*, is also described; but this is considered by some as only an extreme condition of grey hepatisation. The lung is softer, yellowish in colour, and yields to scraping or pressure a quantity of yellow purulent fluid, which is provided by the disintegration of the infiltration filling the air-cells, the leucocytes becoming fatty and granular. A true *abscess*, however, is exceedingly rare as the result of typical acute pneumonia. It is doubtful whether the stage of purulent infiltration is ever reached in cases that recover; it is true, recovery, or *resolution*, is sometimes accompanied by physical signs (*redux crepitation*) which indicate that the exudation is softening into fluid. But many patients get well without such evidence, and

[illegible]

with so little expectoration that the removal of the exudation can only be explained by its absorption; and in few cases is the amount of sputum very considerable. In the rare cases, which are not directly fatal and do not recover, *gangrene* or *chronic pneumonia* may be the result.

The inflammation of the substance of the lung is accompanied, in a larger proportion of cases, by inflammation of the pleura; this often causes pain at the commencement of an attack, may be recognised by friction sound, and may not be again evident during the course of the disease. Pleuritic lymph may be discovered after death, of which there was no evidence during life, or serum, or even pus may be formed in considerable quantity. The double lesion may be spoken of as *pleuro-pneumonia*, but some would only use this name for cases in which the pleurisy was clinically of importance.

Localisation.—Pneumonia is nearly always partial, affecting the base more often than the apex, and the right lung somewhat more often than the left. Beginning at the base of one lung behind, it extends upwards to the apex, as well as forwards; or it may extend downwards from the apex; or commence in the centre and spread upwards and downwards. Occasionally, both lungs are affected; but the disease commonly begins in one earlier than in the other.

Symptoms and Physical Signs.—Shortly stated, the symptoms of a typical pneumonia are pyrexia, beginning suddenly with rigor, continuing with a temperature of 103° or 104°, for five to eight or more days, and ending suddenly or gradually; pain in the side, dyspnœa, cough, expectoration of viscid sputum stained with hæmoglobin; and physical signs indicating consolidation of the lung—viz., dulness, bronchial breathing, bronchophony and increased tactile vibration.

First Stage.—A rigor occurs, in a large proportion of the cases, as the first definite sign of illness; the temperature rises to 102°, 103°, or 104°, and there is well-marked pyrexia, with the usual loss of appetite, furred tongue, and malaise. The symptoms may be at first vague, accompanied perhaps with pain in the head or back; or the implication of the lung may be indicated by shortness of breath and severe pain in the side, attributable to pleurisy. Auscultation at this early period may detect nothing, but there is frequently heard a fine dry crepitation, which has been compared to the noise produced by rubbing between the finger and thumb a lock of hair near the ear; it is mostly heard towards the end of a deep breath, but sometimes during the whole of inspiration; and it is explained by the separation of the walls of the alveoli rendered unnaturally adhesive. The percussion note may at the same time be unaltered; or the physical signs may consist only of a little loss of percussion resonance, with deficiency of vesicular murmur. Even as early as this there may be slight cough, with the characteristic *rusty sputum.* This is brought up as a mass of transparent, airless, jelly-like mucus, of a yellow, orange, russet-brown, or even bright red colour, and extremely viscid, so that it adheres to the side or bottom of the vessel with little or no tendency to flow.

The physical signs of the *second stage,* or stage of consolidation, are often rapidly developed. There is decided dulness over the part of the lung affected. Over the same area there is bronchial breathing, at first perhaps soft and distant, but in a short time, loud, ringing, and metallic. If the patient speaks there is loud bronchophony, the words uttered being often distinctly heard, and apparently shouted up into the stethoscope. The fine crepitation heard as an early sign may still be audible in portions of lung which are being involved by the spreading inflammation; but over areas, which give loud bronchial breathing and bronchophony, crepitations may be heard, somewhat coarser, and generally markedly consonating. Tactile vibration is often, but not always increased. Rarely, instead of dulness, there is a peculiar tympanitic note, or even a cracked-pot sound; and these have been attributed to the presence of a thin layer of still spongy lung between the hepatised portion and the surface. During this development of the physical signs the patient is almost necessarily confined to his bed; he is commonly obliged to sit up; his cheeks and forehead are flushed, with perhaps a slight tinge of jaundice; his eyes are bright, and show a vivid consciousness of his distress; his breathing is quick and shallow rather than laboured, and the respiration may rise to 40, 50, 70, or even 80 in the minute. The pulse is quickened, but not in proportion to the respiration; it may be 100 to 120, or somewhat higher; thus the

FIG. 18.

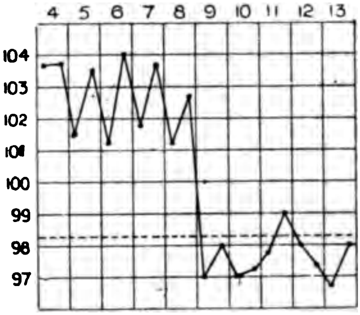

Chart of a Case of Pneumonia, with Crisis on the Eighth Day.

pulse-respiration ratio is altered from the normal 3 : 1 or 4 : 1 to 2 : 1 or 1½ : 1. The temperature is maintained generally at a high level, 103° to 105°, with little variation; and the skin is dry, and gives a sense of pungent heat to the hand placed on it. The cough, which is usually, though not always, present, is not very frequent, is hard, dry, and often painful; and the viscid, rusty sputum is brought up with difficulty. The urine is scanty, high-coloured, acid, and deposits urates; the chlorides are much diminished and may be absent, and there is not infrequently a small quantity of albumen. The patient often retains his consciousness entirely, or may wander a little at night.

The general condition of the patient continues very much the same for some days, or more often there is an increase in the severity of the symptoms. The pulse and respiration are quicker, the temperature continues higher, the tongue becomes drier and browner, and the delirium at night more decided. The physical signs are generally observed to alter from day to day, indicating the spread of the consolidating process, so that crepitation and bronchial breathing extend higher and higher up the chest, until the apex is involved, and the physical signs may be apparent in front under the clavicle.

Later Stages.—One cannot distinguish, clinically, the stage of grey from that of red hepatisation. The stage of *resolution* can be recognised by many indications.

When the illness is apparently at its worst, improvement takes place, in many cases quite suddenly. On the sixth, seventh, or eighth day, in a large proportion of cases, the temperature, the pulse, and respiration fall, in the course of twelve or eighteen hours, nearly to their normal limits, the tongue becomes moist; and the patient feels himself in all respects better. This *crisis* may be accompanied with profuse sweating, or with diarrhœa. In more than half the cases the fever ends more gradually.

The pulse and respiration then become nearly normal, the physical signs quickly or more slowly clear up, the bronchial breathing becomes fainter, and the dulness less marked. In this stage there is often heard a well-marked crepitation, louder coarser, and moister than the early pneumonic sound; it is called *redux crepitation*. The sputum also changes, losing its characteristic tinge, becoming yellowish or greenish, muco-purulent, and at the same time less viscid.

In fatal cases, death occurs from failure of the heart, or from œdema of the hitherto unaffected lung, or from both combined. All the symptoms are aggravated—the respirations are increased in frequency; the pulse is quick, small, and feeble, the face becomes cyanosed; the tongue is dry, brown, and cracked; delirium is more or less continuous, and muttering and coma gradually supervene. On auscultation, loud, coarse râles are heard on both sides of the chest. As the patient becomes feebler, the temperature falls, the skin becomes cold and is bathed in profuse perspiration. Death commonly takes place during the height of the illness, between the fifth and the tenth days. Occasionally, however, a pneumonia runs a fatal course in two or three days.

Complications.—*Pleurisy*, with formation of lymph or serum, is the most frequent. *Empyema* and occasionally *pericarditis* also occur. In a small number of cases there is pronounced *jaundice*; a faint icteric tinge is more common. The cause of jaundice in pneumonia is not clear; it is not due simply to extension, since it occurs both in apical and in left-side pneumonia. Another somewhat rare complication is *parotitis*, which may go on to suppuration.

Diagnosis.—In the early stages of rigor and high fever pneumonia may be indistinguishable from other *acute illnesses*, such as typhoid, scarlatina, or small-pox. Frequently the pain or distress in one side of the chest will indicate acute disease there, and some modification of the breath-sounds at one spot, followed by dulness, bronchial breathing, and bronchophony, will show the nature of the illness. But the pain may be low in the back, and strongly suggestive of variola. In other cases a short cough, with expectoration of rusty sputum, will occur before the development of the physical signs. These last may, indeed, be delayed for five or six, or even ten days, and then the diagnosis may have to be made from the absence of rashes characteristic of the exanthemata, from the rapidity of respiration out of proportion to the pulse, from the flushed face and bright eye, from the characteristic sputum, or from the presence of herpes about the mouth—an uncertain guide.

When physical signs appear, it has to be determined whether pneumonia or *pleuritic effusion* is present, or a combination of both. The diagnosis of these two conditions from one another will be dealt with under Pleurisy; it will be sufficient to say here that pleuritic effusion, though often accompanied by bronchial breathing, causes more absolute dulness than pneumonia, and weakens or abolishes tactile vibration. When they co-exist the pneumonia is often masked by the physical signs of the pleuritic effusion which lies over it, whereas the pneumonia may be signalised by the rusty sputum, and the constitutional symptoms, which are commonly more pronounced than those of pleurisy. Pneumonia very rarely goes on to a chronic condition, and if physical signs supposed to be due to it persist for weeks with continued pyrexia, the presence of pleuritic effusion, serous or purulent, is highly probable.

Prognosis.—The mortality of pneumonia is about 17 per cent. The disease is more fatal to the intemperate, and to those who have been insufficiently fed. Apart from these considerations, it is difficult at the onset of a case to say what the termination will be. Early or violent delirium, failing pulse, cyanosis, the rapid implication of the whole of one lung, the spread of the disease to, or the occurrence of œdema in, the other lung, are all symptoms of bad augury. As already stated, recovery from acute pneumonia is generally complete, and only rarely does a chronic pneumonia directly supervene upon it.

Treatment.—The patient of necessity takes to his bed, and generally in the height of the disease requires to be supported in a semi-recumbent position by means of pillows or bed-rest. The diet should consist of milk and beef-tea, or mutton broth administered in small quantities, frequently. It is almost certain that no drug has any direct influence upon the inflammatory process, and the idea of cutting short the disease, which formerly prompted the use of venesection, mercury, antimony, and more recently the employment of aconite, is now practically abandoned. In early stages the bowels should be opened, and a free action of the skin should be encouraged by the use of acetate or citrate of ammonium, with small doses of Dover's powder. This last will relieve the pleuritic pain, or opium may be more frequently given in small doses (3 to 5 minims of tincture) with the saline. Linseed meal poultices applied over the affected side of the chest, and changed every two hours, often give great relief; they may be sprinkled with mustard, or hot flannels wrung out of turpentine may be applied until they produce decided reddening of the skin, but it is doubtful how much value this form of counter-irritation has. On the other hand, there is an increasing amount of testimony in favour of ice-applications (ice-bags, or pieces of ice between layers of flannel), which relieve the sense of tension, tend to reduce temperature, and are liked by the patients. In mild cases this may be all that is required, but in the severer cases delirium and increasing prostration will have to be met. For the former chloral and potassium bromide may be employed; but chloral must be given with caution where there is much dyspnœa, from its depressing effect upon the heart and respiration. For the same reason, morphia must be sparingly used in the later stages. Stimulants are probably our most important aids under the circumstances, since the delirium commonly coincides with a quick feeble pulse, and other evidences of cardiac failure. Brandy may then be given to the extent of 4, 6, or 8 ounces daily, and ammonium carbonate in doses of 5 or 7 grains every three or four hours. Digitalis or strychnine in small doses may be added to give power to the heart, or quinine in 2 to 5 grain doses for its general tonic effect. Inhalations of oxygen are sometimes of great value in urgent cases. When once the crisis is past, and the temperature has fallen to the normal, the treatment requires simply to be directed to the strengthening of the patient by the administration of quinine and other tonics; since the sequelæ of acute pneumonia are few and infrequent.

BRONCHO-PNEUMONIA.

(*Catarrhal Pneumonia. Lobular Pneumonia.*)

Ætiology.—This form of pneumonia occurs most often in children under five years of age. It often results from the extension of bronchitis to the air-vesicles; it is a frequent complication of measles and whooping-cough; and also occurs after other infectious diseases (diphtheria, scarlet fever). Children habitually breathing an impure air are thought to be more liable to broncho-pneumonia, and it is probable that rickets, by weakening the thoracic movements, also predisposes to it. Lobular pneumonia occurs in adults from inhalation of foreign particles into the lungs, and as a part of septic processes—*e.g.*, pyæmia.

The micro-organisms found in broncho-pneumonia are:—the pneumococcus, streptococcus pyogenes, staphylococcus albus and aureus, and Friedländer's bacillus pneumoniæ. Streptococci are more frequent and pneumococci less frequent than in croupous pneumonia, and the former are more frequent in the cases which are secondary to bronchitis.

Morbid Anatomy.—In broncho-pneumonia the consolidation is scattered throughout the lung in the form of nodules, mostly separate, but sometimes aggregated closely together, so as to form larger masses, but even then still to be distinguished by the eye from each other. On section they are seen as patches, more or less rounded in form, about the size of a pea, reddish-brown, grey, or greyish-yellow, with a less marked granular appearance than in ordinary pneumonia, slightly raised above the surface, solid to the touch, yet soft, friable, and yielding some corpuscular fluid on squeezing. Under the microscope, the contents of the alveoli are seen to be mostly large cells, of an epithelial type, the result of proliferation of the cells lining the alveoli; but inflammatory products from the finer tubes can also be recognised, which have been sucked into the alveoli by the inspiratory efforts. Patches of collapsed lung accompany the consolidation; and where the patches of consolidation reach the surface, they generally give rise to some pleurisy.

Symptoms and Physical Signs.—The former are cough, dyspnœa, and pyrexia; the latter vary with the extent and position of the separate lesions. If the child has already a cough, with rhonchi and râles over the chest, from a preceding bronchitis, the implication of the alveoli may be indicated by a rise of temperature to 102° or 103°, by the cough becoming short, dry, and painful, and by the râles becoming more abundant, and taking on a consonating character. When a sufficient number of consolidated lobules are aggregated together, patches of dulness, with bronchial breathing, and bronchophony, are found; but sometimes the chest remains resonant, and the breath-sounds are only masked by the rhonchi and râles. Young children swallow their sputum, but in those who can be made to expectorate, it consists of mucus with or without streaks of blood. Exceptionally, there may be free hæmoptysis.

The course of the disease is not so definite as in croupous pneumonia. It may end in a week, but often goes on for three or four weeks, or even more. The temperature is generally remittent, or even intermittent, with excessive oscillations of 3, 4, or 5 degrees between morning and evening (*see* Fig. 19). The breathing is rapid and shallow, and is effected in a manner which is often valuable in diagnosis. It has been called *pneumonic breathing*, though it occurs also in pleural effusion; there is a quick inspiration, the breath is held for half a second, expiration then occurs with a grunt, and inspiration again follows without an interval. The lower intercostal spaces are depressed. There is much cough, the face is flushed; or in severer cases pale and livid. The pulse is quick and small. Delirium is often present. The physical signs frequently alter in the course of the illness, indicating the clearing up of disease at one part, and fresh outbreaks in others; and the disease often attacks both lungs. Recovery is mostly gradual, and not sudden, as in croupous pneumonia. In fatal cases convulsions may precede death.

FIG. 19.

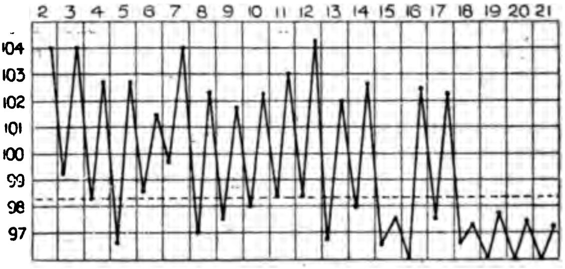

Chart of a Case of Broncho-Pneumonia.

Sometimes, however, broncho-pneumonia is clinically almost identical with the acute pneumonia of adults; it begins suddenly, runs a short course, terminating in crisis, and the physical signs indicate consolidation of a lobe or localised portion of the lung. In such cases, nevertheless, the lesions present *post mortem* a lobular distribution and catarrhal character.

Diagnosis.—Broncho-pneumonia may be confounded in its early stages with other *acute illnesses*, characterised by high fever, such as typhoid fever; and the liability of children to marked cerebral symptoms, from any acute illness, may lead to a diagnosis of *meningitis*. The preceding bronchitis, and the greater predominance of the chest symptoms, may assist; but an opinion may have to be suspended for a few days. Long-continued broncho-pneumonia may give rise to a suspicion of *tuberculosis*, in which high fever, universally scattered râles, lividity, and cough are prominent symptoms. In *capillary bronchitis* there are dyspnœa, lividity, and râles, but the râles are often confined to the bases; there is no bronchial breathing, and expectoration, if present, is purulent. The diagnosis from *croupous pneumonia* is often difficult in children. When the consolidation affects a large area continuously on one side only, when the illness begins abruptly, has a temperature without much remission, and ends with crisis, it is commonly regarded as croupous. But it has been shown that broncho-pneumonia may have a lobar distribution; and it is stated that a pneumococcal pneumonia—that is, a croupous pneumonia—takes on a lobular distribution in children (West). Bacteriological examinations are difficult in children, who supply no sputa. Hence, the course and duration of the fever remain as the chief guides.

Prognosis.—Though this form of pneumonia is much more fatal than the croupous variety, the prognosis must depend upon the general progress of the symptoms. Cases that are apparently desperate often recover, and an unfavourable opinion should be given with some caution. In the broncho-pneumonia of old people, and in those due to the inhalation of particles, the prognosis is more grave.

Treatment.—The treatment may be conducted on the same general principles as in the case of ordinary pneumonia. A jacket-poultice may be applied all round the chest, and a steam-kettle may be used. But here, again, very good results have been obtained with ice applications to the chest, even in quite young children. Expectorants, such as ammonium carbonate 1 grain, and vinum ipecacuanhæ 2 minims, should be given every four hours. Severe cases generally require stimulants to be administered rather freely—*e.g.*, for a child three or four years old, 20 minims of brandy every hour.

Abscess of the Lung.

Apart from the suppurating cavities determined by tubercular disease (*see* Phthisis) abscess of the lung is comparatively rare. It has already been mentioned as a result of pyæmia, and of acute pneumonia. It may be determined also by foreign bodies entering the bronchi; and new growths, such as cancer, and probably spyhilitic gumma, may occasionally break down and suppurate. As it is always a secondary occurrence, its symptoms are grafted upon those of the preceding condition. The physical signs are not likely to be distinguishable from consolidation, until the abscess has burst, and discharged pus; and this may occur suddenly. The usual signs of a cavity, such as tympanitic resonance, cavernous or amphoric breathing, metallic râles, and pectoriloquy may then be heard. The multiple small abscesses of pyæmia are not recognisable as cavities: indeed, their presence is usually masked by the surrounding consolidation, or pleuritic effusion; and death may have taken place almost before the abscess has softened down into fluid pus.

Treatment.—An abscess after acute pneumonia has occasionally been assisted by surgery; otherwise the patient must be supported by nourishing food, quinine, iron, &c.

CHRONIC PNEUMONIA.

(*Cirrhosis of the Lung.*)

Ætiology.—This form of lung disease is comparatively rare; the great majority of chronic inflammations of the lung-tissue being associated with tubercle, and included under the term phthisis. The cases in which chronic pneumonia is independent of tubercle, and to which the names chronic pneumonia and cirrhosis of the lung are given, have arisen only in a few instances from a preceding acute croupous pneumonia; but catarrhal pneumonia has been a more frequent antecedent. Chronic bronchitis, bronchiectasis, and chronic dry pleurisy, appear to be the causes in other instances.

An important class of cases in which chronic pneumonia succeeds to bronchitis, is that occurring in various manufactories and known as *pneumono-koniosis*. Here the repeated inhalation of an atmosphere laden with the dust of coal, metal, stone, cotton-fibre, fluff, &c., provides a life-long source of irritation. The disease has received different names according to the particular irritant concerned—*anthracosis* (coal dust), *siderosis* (steel), *silicosis* (stone). Fagge held the view that there were cases of phthisis, induced by the bronchial irritation; but though some patients no doubt become tubercular, this does not seem to be the case with all.

Morbid Anatomy.—The characteristic feature of the lung affected with chronic pneumonia is the excessive development of fibrous tissue in its substance. At first the lung is traversed with bands of fibrous tissue, arising in the interlobular septa; in late stages the whole lung may be converted into a dense mass of fibrous tissue, of various shades of grey, from the presence of pigment, tough in consistence, and creaking under the knife. In cases of dust disease, the lung is coloured black, red or grey, according to the nature and quantity of the particles inhaled. With the growth of the fibrous tissue contraction takes place, and the lung may be reduced to two-thirds or half its natural size; the excavations commonly occur partly from dilatation of bronchial tubes, and partly from ulcerative processes in the lung-substance. Nearly all cases are accompanied by a chronic pleurisy, and the lung is fixed to the chest by a thick fibrous layer. The contraction of the lung leads to displacement of organs, and, as usually only one side is affected, the mediastinum is pulled in that direction.

Symptoms and Course.—The disease is essentially chronic, and patients in whom it is recognised have generally complained for some months or years. They are short of breath, and have cough and expectoration, which vary with the extent of the cavities in the lungs. When there are large cavities, or much dilated tubes, the cough may be paroxysmal, with abundant and perhaps fœtid expectoration. The patient is often thin, but may be well nourished, and is at any rate for a time free from the fever, night-sweating, and general constitutional disturbance observed in tubercular phthisis. Hæmoptysis is, however, often present. Some of the local conditions have been already indicated. The disease is generally unilateral, the corresponding side of the chest is retracted, the shoulder depressed, and the angle of the scapula tilted outwards; the impulse of the heart is shifted towards the affected side, and the healthy side of the chest is hyper-resonant. The affected side expands but little; it is dull on percussion. The respiratory sounds are feeble or distant; and if large cavities or much dilated tubes are present, the breathing may be hollow or tubular, with metallic or bubbling râles. Such cavities are more often situate about the middle level of the lung, than at the summit, as in phthisis. Tactile vocal fremitus is commonly diminished. Thickening or *clubbing* of the finger-ends (p. 464) is often pronounced. For a time the compensatory action of the healthy lung suffices to maintain efficient oxygenation of the blood, but in time the right side of the heart becomes dilated, and cyanosis and dropsy ensue.

Diagnosis.—The condition has to be distinguished from ordinary phthisis, from chronic pleurisy with effusion, and from malignant growth in the chest. From *phthisis* the absence of fever and constitutional disturbance is the chief distinguishing feature; the disease is mostly rigidly unilateral, whereas phthisis rarely reaches an advanced stage in one lung without affecting the other; and tubercle-bacilli are not found in the sputum. *Pleuritic effusion* of old standing with retracted chest may closely resemble the fibroid lung, and exploration with a needle may be required to clear up the diagnosis. Pleuritic fluid, whether simple or purulent, will be generally accompanied by feverishness, and, on the other hand, crepitation and râles will be much in favour of chronic pneumonia. *Malignant disease* may be excluded, according to the late Sir Andrew Clark, if there be no cachectic appearance, no irregular retraction of the chest, no pains or signs of pressure with extensive consolidation, and no extension of dulness beyond the normal limits of the lungs.

Prognosis is ultimately bad, but the course may be very slow, extending over ten or fifteen years. Death may take place from failure of the right heart, or from the gradually increasing exhaustion which follows profuse discharge.

Treatment.—The patient should be placed under the best possible climatic and hygienic conditions. He should have bracing air in the summer, but a warm climate in the winter; avoid exposure to chills at all times; and have nourishing diet and tonics, such as quinine, iron, and cod-liver oil. Cough, expectoration, and other symptoms, should be treated as they arise, in the same manner as directed under Phthisis and Bronchiectasis.

GANGRENE OF THE LUNG.

This is a comparatively rare disorder, which may arise, however, under a variety of circumstances. It is one of the terminations of acute croupous pneumonia, and occurs sometimes in phthisis; it may result from the invasion of the lung by contiguous diseases like cancer of the œsophagus, abscesses and suppurating hydatid cysts, and from the pressure of aneurysm on the root of the lung; as a result of foreign bodies lodged in the bronchus, and from the presence of secretions retained in dilated tubes; from the inhalation into the lung of particles from septic diseases in the mouth, throat, or larynx, such as cancer of the tongue or larynx, sloughing of the tonsils, and diphtheria; from particles of food drawn into the lung by accident, or during vomiting, especially in those who are drunk, insane, comatose, or suffering from laryngeal paralysis. Gangrene of the lung is also sometimes caused by septic particles brought to it by the blood-vessels, as in various pyæmic processes, after otitis, bed-sores, puerperal disorders, &c. Practically all these causes are frequently in operation, without causing gangrene; this is more likely to occur under the influence of certain predisposing conditions—namely, marasmus, old age, intemperance, and diabetes. The immediate factors in the production of gangrene appear to be presence of septic organisms, and conditions of low general vitality, or the existence of local unfavourable conditions such as aneurysm or cancer pressing on the vessels at the root of the lung, or embolism of a pulmonary artery.

Morbid Anatomy.—The affected portion of lung is of a dirty, greenish-brown, or black colour, soft, readily breaking down, or even diffluent; and often emitting an offensive odour. It is generally surrounded by consolidated pneumonic tissue, into which it may gradually pass, or from which it may be more or less sharply marked off by a line of demarcation; the former condition is, according to Fagge, only an earlier stage of the latter, but previous writers have mostly described them as two separate forms, diffuse and circumscribed. The gangrenous tissue may break down, and be expectorated, so as to leave a cavity with ragged, shreddy walls; and occasionally such a cavity opens into the pleural sac and causes pyo-pneumothorax.

Symptoms.—Gangrene of the lung occurring, as it often does, as a secondary lesion, just before death, may be readily overlooked. On the other hand, its symptoms may stand alone, or overshadow those of the primary lesion. Fœtid expectoration and fœtid odour of the breath are the most prominent. The latter may be very penetrating, it is carried to a great distance, and makes it almost impossible for other persons to live in the same room with the patient. The sputum is dirty grey, or greenish-brown, or black, from altered blood; and either fragments of gangrenous lung-tissue are found, or the microscope detects the typical elastic fibres (p. 460). Occasionally hæmoptysis takes place. Cough, pain in the side, and irregular, and mostly intermittent, pyrexia are also present. The physical signs are those of consolidation and cavity proportionate to the extent of lung diseased—viz., dulness, bronchial or cavernous breathing, bronchophony, and medium or coarse râles; but their value in diagnosis must depend a good deal on the preceding disease, if any. The onset of gangrene may be soon followed by prostration, with quick small pulse, dry tongue, and death at no great distance of time. Some cases, however, last for months or years with much variation in the intensity of the symptoms, but without escaping a fatal termination. And in a few cases, with probably a very small patch of gangrene, recovery actually takes place.

Treatment.—This is similar to that of fœtid bronchitis. Antiseptic inhalations (creosote, carbolic acid, eucalyptus oil, thymol) or the antiseptic respirator should be frequently used. The strength should be supported by quinine, cinchona, iron, ammonia, good food, and sufficient stimulants. A gangrenous cavity may sometimes be amenable to the surgical treatment of antiseptic incision and drainage.

PHTHISIS.

(*Pulmonary Tuberculosis. Consumption.*)

By the term phthisis (φθίω, I waste) is now meant a destructive disease of the lung, brought about by the invasion of the *bacillus tuberculosis* (*see* p. 136). The essential features of the disease are the formation of *tubercles* as the result of the irritation of the bacilli; the occurrence of pneumonic processes in connection with the tubercles; the consolidation of portions of the lung; the subsequent breaking-down of tubercular and pneumonic areas into cavities; suppuration and the discharge of *débris* of lung-tissue by expectoration; and general constitutional disturbance of varying severity. The majority of the complications and associated lesions can be explained by the spread of the process in the lungs, or by the co-existence of the tubercular lesions in other organs.

Ætiology.—Accepting the bacillary origin of phthisis, we must suppose that in a community where phthisis is prevalent every one is brought into contact, from time to time, with the bacillus; but that tubercle only develops when the bacillus meets with a condition of the tissues favourable to its growth. The question of ætiology resolves itself into the methods of introduction of the bacillus into the body, and the conditions which will secure its persistence and multiplication. At present the facts bearing upon the latter half of the question have the greater importance. Nothing seems to be better established than the truth of the hereditary transmission of phthisis. The disease frequently occurs in the offspring of a phthisical parent; and if both parents are phthisical, or if one has the disease, and the other comes of phthisical stock, the tendency on the part of the children is greater. Nevertheless, the phthisical patients, who give a history of phthisis in their parents, form only about 30 per cent. of the whole number. There are good grounds for believing that in extremely rare instances the tubercle bacilli or their spores are actually transmitted from either parent to the fœtus, and this happens certainly in animals. But in nearly all cases what is handed down is a tendency only, and the child is not born with phthisis or any tubercular lesion. The invasion of tubercle may be successful at any time of life from two or three months to forty or fifty years; as a rule, however, the disease commences before the age of twenty-five, and commonly rather sooner in males than in females.

This tendency to suffer from tubercular lesions, pulmonary or otherwise, was known as the tubercular "diathesis," and was believed to be revealed by certain characteristics of physical growth and mental capacity. A dark, beautiful, or tubercular type, and a fair, coarse skinned, or scrofulous type were distinguished. But numbers of phthisical individuals are of quite indifferent build and appearance, who could not be classed with either type, and the most that can be said of the supposed diathesis, is that it perhaps represents some delicacy of tissue, which is little resistant to the invasion of the bacillus.

A disposition to phthisis may be acquired by any circumstance, or combination of circumstances, which seriously lowers the vitality of the body, whether this be a deficient supply of food and fresh air, or prolonged debilitating illnesses, or special toxic influences. The most frequent of these are:—(1) Overcrowding, and deficient ventilation, working in close rooms in the fumes of gas, &c.; (2) deficient supply of food, which frequently co-operates with the first cause, as well as the next; (3) exhausting work, in association with the preceding; (4) frequent child-bearing in women, and the exhausting drain of lactation; (5) exposure to wet and damp: Buchanan showed that amongst communities living on damp and imperfectly drained soils there was an undue proportion of deaths from phthisis and lung diseases; (6) enteric fever; (7) excessive indulgence in alcoholic drink; (8) diabetes mellitus; (9) syphilitic cachexia.

Local lesions situated in the lungs themselves may prepare the soil for the growth of tubercle. It is a common belief that repeated catarrhs, or even one neglected catarrh, will cause phthisis; and though there is little doubt that often the cough, which is the first indication of tubercular deposit, is mistaken for an independent catarrh, a prolonged catarrhal bronchitis, or a catarrhal pneumonia, may lower the resistance of the lungs, and so lead, with other favouring circumstances, to tubercular deposit. This is especially the case with the pulmonary inflammations following measles and whooping-cough, and probably a croupous pneumonia may sometimes act in the same way. As already stated (p. 448) tubercular disease not infrequently supervenes upon the bronchial irritation and chronic pneumonia of workers in certain manufactories.

If we now consider the means by which the bacilli are introduced into the system, we must admit the possibility that phthisis can be communicated directly or indirectly from man to man. We have already seen that this may be by means of a wound. In the chronic *post-mortem* lesion, known as *verruca necrogenica*, tubercle-bacilli have been found, and unmistakable tubercular infection has resulted in some such cases. Here it must be presumed that the person has accidentally infected himself from a patient dead of tubercular disease. But these are rare instances, and in most cases of phthisis we must suppose that the bacillus gains access to the lungs by means of the inspired air.

That simple contact with phthisical patients is not sufficient is shown by the daily experience of medical men, and the immunity of the resident medical officers and nurses of the Brompton Hospital for Consumption has often been quoted in support of this. But there are strong grounds for believing that the disease is occasionally transmitted from husband to wife, or has passed between brothers and sisters, or others living together. The researches of Cornet show that the chief agent in the diffusion of the bacillus is not the air expired by the phthisical patient; but the sputum, which, as is well known, may be loaded with the specific micro-organisms. If this is repeatedly ejected on to the floor of a room and allowed to dry, or if quantities of it dry upon handkerchiefs, the air of the room may at length be sufficiently impregnated to become dangerous to healthy people breathing it. From the floor and walls of rooms formerly tenanted by phthisical people, Cornet obtained bacilli, by the inoculation of which he produced tubercular disease in healthy animals. If this view of the situation is correct, it helps to explain the deadly influence of deficient ventilation in workshops, manufactories, barracks, and similar institutions, as well as some of the instances of fatal prevalence in families. But in the majority of cases it is difficult to ascertain the source of contagion, whether invasion is by the lungs or elsewhere, from the slowness of development of tubercle as compared with other infectious diseases.

There is some evidence that tubercular infection may be due to the ingestion of the flesh or milk of tuberculous animals, but it seems probable that the disease would then begin in the abdomen rather than in the lungs. Pulmonary phthisis might be a secondary result of this, as it may be a sequel of tubercular lesions in any part of the body (*see* p. 457).

Morbid Anatomy.—The nature and structure of tubercles have been already described (p. 135). They occur in a most typical way in the lungs, with their adenoid structure, giant-cells, and bacilli, and their tendency to caseate and break down. They develop first in the interstitial tissues, especially in the alveolar walls, and in the peribronchial, perivascular, and subpleural tissues. From the alveolar wall they invade the air-vesicles, and may seem afterwards to be situate in them; and the tubercles situate in the walls of the small bronchi will project into and narrow their calibre. This simple growth of tubercle constitutes the *first stage* of phthisis.

After a time the tubercles are succeeded by pneumonic processes, of catarrhal and, less often, croupous kind, by which small areas of consolidation are produced, which may be more or less intermixed with masses of tubercles now becoming altered by caseation from the grey to the yellow variety. This is the *second stage*, or stage of *consolidation*.

The *third stage* is that of *excavation*, or the formation of *cavities* or *vomicæ*. It arises by the breaking-down and disintegration of cheesy tubercles, and of pneumonic lung. By a mixed process of caseation and suppuration the cavities become larger and larger; adjacent cavities run into one another, and ultimately the lung may be extensively hollowed out. In their earlier stages the walls are often formed of caseous deposit; but in old vomicæ they are quite smooth, like mucous membrane. They are often traversed by bands, or trabeculæ, which contain pulmonary vessels. The vessels resist the destructive process; whereas the bronchi are generally ulcerated in proportion as the cavities enlarge, and into each cavity one or more bronchi open, often by an aperture much narrower than the calibre of the tube above. The contents of vomicæ are caseous matter, *débris* of lung-tissue, and pus. The latter predominates in the older cavities; the quantity is very variable, and it may be so small, under certain circumstances, that no expectoration takes place for considerable periods. It is only rarely that decided putrefaction takes place in phthisical cavities.

But in the majority of cases this process of destruction does not have full play. The inflammatory changes present varying changes of activity in different cases; and the mischief may be stopped for long periods one or more times in its course, or may even become abortive at an early date, and go no farther. The development of *fibrous*, *connective*, or *cicatricial* tissue is the important agent here. It is rarely absent in any but the acutest cases, and in the chronic cases it forms a large proportion of the remaining tissue of the diseased lung. In the consolidated lung there are numerous bands running in the course of the interlobular septa, surrounding the bronchi, the blood-vessels, and the cavities, and forming a dense layer under the visceral pleura. The fibrous tissue is frequently deeply pigmented, and is mixed here and there with caseous masses. By its contraction it tends to diminish the size of the cavities, and opposes some resistance to destructive processes, and in some favourable cases a small deposit of tubercle may be ultimately converted entirely into a mass of pigmented fibrous tissue, which, indeed, replaces a similar amount of healthy lung, but is otherwise harmless. In such cicatrices it is not uncommon to find calcareous particles, from the deposit of lime salts in the caseous material. Another change accompanying such a cicatrix is a local emphysema, which has been called *compensatory emphysema*, since it serves to fill the space left by the contracted portion of the lung.

When the process is advanced the pleura seldom escapes. The formation of tubercle in the pleura is not common; but inflammation of the membrane is the result of the extension of the pulmonary change, whether of consolidation or excavation, to the surface. The *pleurisy* is often chronic, or subacute; if acute, the area invaded at one time is but small. The final result is the formation of a thick layer of membrane over the affected portion of lung, commonly uniting the organ firmly to the wall of the chest.

This adhesion of the lung has an important protective influence, for, if the process of excavation advances to the surface at a point which is not adherent, the vomica may ulcerate through, and discharge its contents into the pleural cavity, leading, on the one hand, to an acute pleurisy, generally of the purulent variety—*empyema*, or pyothorax; and on the other, to the entrance of air into the pleural sac—*pneumothorax.*

Another important result of the destruction of tissue is *hæmorrhage:* in earlier stages this follows from congestion alone; in later stages the vessel walls are directly invaded by tubercle, and hence may be eroded; or the vessel wall, weakened by tubercle, dilates so as to form an aneurysm, which may reach the size of a pea or bean, and ultimately give way at its thinnest part.

Situation of the Lesions.—As already indicated, the above changes follow one another with very variable rapidity, and the spread of the disease through the lung is equally irregular as to absolute time. But the situation of the lesions, and the order of their invasion, are subject to some very constant rules.

The first deposit of tubercle takes place at the apex of the upper lobe; and fresh deposits occur at intervals of weeks, months, or years, lower and lower down. This invasion of fresh parts of the lung no doubt takes place not by the vessels as in a miliary tuberculosis but through the bronchi, infective particles being inhaled, and starting fresh centres of disease. By the time that tubercle forms at the lower levels, the first lesion may have reached the second stage, or stage of consolidation, and later on, when tubercle is being deposited towards the base, the middle part of the lung will have got into the second stage, and the apex into the stage of excavation.

Thus one lung may, and frequently does, present all three stages—the apex with cavities; below this, consolidation, with fibrous tissue, pneumonic patches, and caseous tubercle; below this, mostly scattered grey tubercles, with perhaps some congested lung tissue; and below this, finally, some quite healthy lung.

Again, the progress of the disease, while unequal in any one lung, is unequal in the organs on the two sides. As a rule, before a patient dies of phthisis, both lungs are affected, but rarely to the same extent; a large area is commonly involved on one side, before the other is attacked; and so in an advanced case it is common to find the most extensive disease at one apex, and the most healthy lung, or the only healthy lung, at the opposite base. The law of the extension of lesions from apex to base may be supplemented by the rule that the apex of the lower lobe is often invaded soon after the apex of the upper lobe, and before the lower part of the upper lobe: and in testing the truth of this observation clinically, it must be remembered that the lower lobe occupies the greater part of the back of the chest, reaching as high as the third dorsal spine, or the spine of the scapula, and that the greater part of the front of the chest corresponds to the upper lobe. Kingston Fowler has further defined the points at which invasion first occurs as follows:—In the upper lobe the disease begins at a point an inch to an inch and a half below the top of the lung, and rather nearer to the posterior and external borders; from this point the disease often extends downwards, by fresh scattered deposits along the anterior aspect of the upper lobe, about three-quarters of an inch from its margin. A less common situation for the first deposit corresponds on the chest wall with the first and second interspaces below the outer third of the clavicle; and from this point the lesions extend downwards and backwards. In the lower lobe the usual seat of invasion is about an inch to an inch and a half below the upper and posterior extremity, and about the same distance from its posterior border; and extension takes place backwards towards the posterior border of the lung, and laterally along the line of the interlobar septum, which position, Fowler points out, corresponds on the chest to the vertebral border of the scapula when the hand of that side is hooked over the opposite shoulder.

Changes in Other Organs.—Phthisis of long duration is commonly associated with lesions of other organs, some of which are of a tubercular nature, while others are of a degenerative kind, but clearly the result of the tubercular cachexia. The most constant are tubercular disease of the larynx; tubercular disease of the intestines; fatty infiltration of the liver; and lardaceous disease of the liver, spleen, kidneys, and intestine.

For a description of these the reader is referred to the sections dealing with the organs concerned.

At any time, also, in the course of phthisis infection of other organs with tubercle may take place, forming an acute general, or miliary, tuberculosis. In this case the healthy remainder of the lungs, the liver, spleen, kidneys, the cerebral and spinal meninges, and perhaps other parts, are invaded with grey tubercles; and death soon follows, either from tubercular meningitis, or, if the meninges are spared, from implication of the lungs, or exhaustion (p. 137). Most of the other tubercular lesions in the body, and many suppurative lesions, are from time to time associated with phthisis, sometimes preceding, sometimes following, the deposit in the lungs: such as caseous or suppurating cervical glands, tubercular disease of the bones, tubercular pyelitis, tubercular peritonitis, scrofulodermia, anal fistulæ, subcutaneous abscesses, &c.

Varieties of Phthisis.—Pulmonary tuberculosis may run a rapid or a slow course. The most familiar is the *chronic tuberculosis*, which lasts from six months to a few years. The description which follows will mainly apply to this. Acute forms of pulmonary tuberculosis occur as *pneumonic phthisis*, and acute miliary tuberculosis, which has been already dealt with (*see* p. 138), and is not generally included under the term phthisis.

Clinical History.—The symptoms which characterise phthisis are the following:—Cough, dyspnœa, purulent expectoration, emaciation, hectic fever, and often hæmoptysis.

The commencement is variable. Many cases begin with cough and expectoration of muco-pus or pus, for which no cause can be given, or which is referred to some chill or exposure. Other cases begin with hæmoptysis or spitting of blood. The patient may have been apparently in good health, when, sometimes after an effort, but quite as often when still, or walking or doing something which involves no strain, a tickling is felt in the throat, the patient coughs, and is surprised and alarmed to find that what he spits is blood. Thereupon he may expectorate a few drachms or an ounce, or even half a pint. This may remain as the only symptom, and an examination of the chest may reveal nothing. But after a time, with or without a fresh loss of blood, cough and expectoration supervene, and the case develops like others. In a small number of cases the first apparent departure from health is an acute pneumonic process in one upper lobe, which only partially clears up, while cough and expectoration persist, and the case takes on all the features of phthisis. Lastly, in some cases indigestion, with loss of appetite, frequent vomiting, and emaciation are prominent symptoms for some time before the special indications of a chest lesion are apparent.

When the disease is once definitely present, it runs its course with different degrees of rapidity; it may be fatal in three or four months, or it may last twelve or fifteen years before finally killing the patient; and in this time its progress will be very unequal, often quiescent for months or a year or two, and then making great strides, with hæmoptysis or much fever. While the more rapid cases are fatal chiefly by the extent of lung involved, the cases of longer duration threaten life by a number of complications, some of which are lesions of the lung itself, such as hæmoptysis, pneumothorax, empyema, and bronchitis; others involve distant organs, such as tubercular meningitis, ulceration of the intestines and diarrhœa, nephritis, and lardaceous disease of the viscera.

But the course of phthisis is not always to a fatal termination, as is shown by both clinical and pathological observation. Early cases, placed under favourable conditions of climate and hygiene, may completely regain their health; and a hæmoptysis, similarly treated, may never recur. The pathological evidence is that in persons killed by accident, or dying of disease unconnected with the lung, it is not uncommon to find cicatricial and pigmented patches, with perhaps cretaceous deposits, in the apices, which can only be regarded as the remains of former tubercles.

Local Symptoms.—These will now be described somewhat more in detail.

Cough.—This is a very common symptom, and generally, though not always, present as long as the disease is in any degree active. It is mostly easy at first, sometimes not much more than a clearing of the throat; it becomes harder and more painful in the later stages; and with extensive cavities it occurs in prolonged attacks, painful to the patient, distressing to those about him, and lasting perhaps more than a minute, until at length some sputum is brought up. With laryngeal complication the cough acquires a hoarse or husky quality.

Expectoration.—In the early stages this is not different from the sputum of bronchitis—that is, it is either simply mucous, or it is muco-purulent; and this is accounted for by the bronchitic processes that frequently accompany phthisis. But sometimes comparatively early, and always in later stages, the sputum becomes purulent, of green or greenish-yellow colour, opaque, and quite free from air-bubbles. If it is very fluid the individual sputa may run together and lose their separate form; but the sputa of phthisis often keep separate long after expectoration, and, from the round, flat shape that they assume in the sputa-vessel, they are called *nummular*, or coin-shaped. This is no doubt due to the accumulation of the secretion in cavities in the lungs, and hence it constantly occurs in phthisis, but may also be present in cases where the cavities are produced by dilated bronchi (bronchiectasis). What especially distinguishes the sputum of phthisis from that of other pulmonary diseases is the presence of blood, of elastic fibres, and of the tubercle-bacillus.

Hæmoptysis.—It has already been mentioned that hæmoptysis may be the first sign of phthisis. The blood is generally bright red and frothy; it is expectorated in variable quantities, and, as a rule, for some hours or days the patient continues to spit pellets of blood which have a darker and darker colour, become gradually less frequent, and then cease entirely. There may at this time be no other sputum. In later stages, when the disease is well established, the muco-purulent or purulent sputum is often streaked or stained with blood. A few streaks in the sputum may proceed from small vessels in the bronchial mucous membrane, but more characteristic of phthisis is the intimate mixture of bright blood with the sputum, or the discharge of pellets of coagulated blood frequently during the day. From time to time may occur more abundant hæmorrhages, like those first described, in which the blood comes up apart from the ordinary secretion; and if a large vessel is ulcerated, or, what is more often the case, if a small aneurysm in a cavity ruptures, several ounces or a pint or two of blood may be discharged within a short time, and rapidly lead to death.

Elastic Fibres from the Lung.—If phthisical sputa be examined with the microscope, there will be found pus, mucus, and blood-corpuscles, drops of myelin, pavement epithelium from the mouth, and alveolar epithelium from the lungs. These may all occur in the sputa from other diseases, but it is said that alveolar epithelium does not occur in ordinary bronchitis in individuals under thirty years of age, so that its presence may sometimes afford useful information. When the destructive stage is reached, fragments of elastic tissue from the walls of the vesicles are mixed up with the expectoration; they may be seen with the microscope on teasing out the little tough nodules which are sometimes found. But the best plan is to boil about an ounce of the sputum with an equal quantity of solution of caustic soda until the whole is perfectly fluid. This requires from twenty to thirty minutes. The liquid is then placed in a conical glass, and the undissolved fragments of resistant elastic fibres are allowed to collect at the bottom, from which they may be removed with a pipette for examination.

Tubercle-Bacilli.—These require to be stained, and to be submitted to a microscopic power of 350 or 400 diameters. There are several methods; the following is Ehrlich's: A microscopic cover-glass is smeared over with a thin layer of sputum, and is passed two or three times through the flame of a spirit lamp to coagulate the albumen. It is then floated, with the sputum downwards, on the staining fluid, prepared as follows:—5 c.c. of anilin are added to 100 c.c. of distilled water, well shaken and filtered: to the filtrate is added a concentrated alcoholic solution of fuchsin, until precipitation commences. After about thirty minutes the cover-glass is taken off, rinsed in water, washed in dilute nitric acid (1 to 2 or 3), and again rinsed in distilled water. It is then put for a minute in a one or two per cent. aqueous solution of methylene blue, and dried and mounted in Canada balsam. The ground is stained blue, and the bacilli red.

Dyspnœa.—Shortness of breath is often early noticed, and becomes very marked as more and more of the lung gets diseased, and so the surface available for aëration of the blood is diminished.

Physical Signs.—These are best considered in reference to the three different stages of phthisis, remembering that all these stages may be present at the same time in the same lung, and that the process is most advanced at one or other apex.

In the *first* stage (tubercular deposit) the physical signs may be very slight, and vary considerably in different cases. The eye or the hand may detect a slight impairment of mobility on the affected side. For this purpose one hand should be laid on either chest just under the clavicle, and the relative movements should be watched during tranquil and during full, yet gentle, respiration. Careful percussion of the apex may give only slight impairment of the note as compared with the opposite side; this may be found just below the clavicle, or on the clavicle, or in the supra-clavicular fossa, or it may be behind, above the scapula. Below the clavicle there may be tenderness on percussion. Auscultation often gives much more certain indications, especially a diminution of the vesicular murmur, and the presence of fine or medium râles during inspiration. Sometimes there is nothing but the deficiency of vesicular murmur, but this, if associated with impaired resonance or mobility, is of much importance. The inspiratory murmur may be irregular, jerky, or wavy—the so-called cog-wheel respiration; or it may be roughened; or the expiratory murmur may be loud and prolonged, assimilating the respiration to bronchial breathing, and this may be associated with an increase of vocal resonance. It is, however, very important to remember that prolonged loud expiratory murmur with loud vocal resonance is not uncommon upon the right side in healthy individuals, and especially in females. And, as a rule, repeated examinations at short intervals are needed before one can with confidence state that there is evidence of phthisis from the physical signs, although cough, expectoration, wasting, and febrile reaction may justify the strongest suspicions. Râles with deficient vesicular murmur are the most trustworthy.

In the *second* stage (consolidation) the physical signs are in many respects similar to those of the second stage of pneumonia. According to the extent of lung involved, there is more or less impairment of mobility of the affected side; and when the progress has not been unusually rapid, there is obvious depression of the supra-clavicular and infra-clavicular regions, caused by contraction of fibrous tissue, or perhaps by the earliest destruction of tissue, producing cavities as yet too small to be recognised by physical signs. On percussion, there is increasing loss of resonance as the case goes on; but the dulness is rarely so absolute as that which occurs over a pleural effusion; and sometimes it has a high-pitched, boxy, or more tympanitic character. On auscultation, bronchial breathing of different qualities and pitch is heard, and the voice and cough are loudly bronchophonic. Râles may or may not be present; they are mostly of sharp clicking, or consonating character.

It is in the *third* stage (excavation), when the disease has existed some time, has seriously involved one lung, or has already attacked the other, that one can best recognise the modifications in the shape of the chest, which may have already commenced in preceding stages. The chest takes on the type of extreme expiration. It is flat, long, and narrow; the shoulders are depressed and sloping; the lower ribs come within a short distance of the crest of the ilium; the upper ribs in front are wide apart, the lower ribs are crowded together, and the costal or epigastric angle is reduced to its smallest size. The nipple tends to lie high in relation to the ribs—for instance, in the third space—while the heart may strike the fifth rib, instead of the fifth space: as if the ribs had glided down between the skin and the viscera. In addition to this general change in the chest, there is retraction of the upper part of the chest on the most affected side, and a corresponding impairment of movement. At this point, which we now suppose to be the seat of cavities, we find that the percussion note is variable. It must be remembered that the cavities form in lung that has first become solid. Now, absolutely solid lung gives dulness on percussion, and a lung entirely hollowed out into one large cavity gives a resonant note; the percussion note over excavated lung must, therefore, vary with the size of the cavity, its nearness to the part of the chest percussed, the amount of consolidation around it, or between it and the point struck, and the degree to which the ribs are fixed by pleuritic adhesions. The note may, therefore, be quite dull, or of tympanitic resonance; more often it is dull to light percussion, and of different degrees of boxy, or high-pitched, half-resonance on a heavier stroke being used. If there is a large cavity in free communication with a bronchial tube, and the patient's mouth be open, percussion will often elicit the *cracked-pot sound* or *bruit de pot fêlé*, which is somewhat, but not exactly, like the sound produced by striking the two clasped and hollowed hands upon the knee to delude children with a hope of pence. The resonance over such a cavity is raised in pitch when the patient opens his mouth (Wintrich); and during inspiration, while it falls in expiration (Friedreich); and the pitch varies with change of position of the patient (Gerhardt). On auscultation over cavities, one may obtain hollow bronchial, or cavernous or amphoric breathing, according to the varying degree of excavation and condensation around. It is only truly amphoric when the cavity is very large indeed. The vocal resonance may be simply increased (bronchophony), or it may be pectoriloquous as well, the whispered voice being transmitted with unusual distinctness; or pectoriloquy may be present alone. In extensive cavities, when the patient speaks, there is heard, in addition to the loud vocal resonance, a kind of *whispering echo* of the same, apparently produced by reverberation from the walls of the cavity. Bubbling râles of large size are often heard in cavities, and the peculiar phenomenon known as metallic tinkling. It must be remembered that the auscultatory sounds are best brought out on deep inspiration; that they may all be absent if, and as long as, the bronchial tube in connection with the cavity is blocked; and that a cavity probably does not give distinctive signs as compared with consolidation, unless it has reached a large size, about that of a walnut. *Port-tussive suction* is a high-pitched sound heard during the inspiration following a cough, due perhaps to elastic recoil of the walls of the cavity.

In cases of long standing, in which the left lung is mainly affected, the contraction of that organ allows the heart to come more fully into contact with the chest-wall, and in the second left intercostal space may be observed a pulsation, due to the conus arteriosus of the right ventricle; the closure of the pulmonary valves may be then felt with the finger, and the second sound is unusually accentuated, or rather it is heard with greater distinctness than is normal.

General Symptoms.—The more important are pyrexia, with night sweating; loss of flesh and strength; anæmia; and the evidences of imperfect aëration of the blood.

Pyrexia.—From the earliest days of phthisis, fever may be present, but it generally bears some relation to the activity of tubercular and pneumonic processes in the lung, so that if the mischief becomes inactive from time to time, the fever may for a corresponding time be absent; but it is often present continuously for months. The temperature is commonly higher in the evening than in the morning, and is either remittent or intermittent in type. In the former it may be 99° to 100° in the morning, and 102° or 103° in the evening; in the latter it is 98.4° or even lower in the morning, and reaches 100° to 103° in the evening. The lesser degrees of fever are often not appreciated by the patient; the higher are accompanied with the discomfort and malaise common in pyrexia, and especially in the advanced stages of the disease, with profuse sweats, and even slight chill before the sweating. Actual rigors, however, are exceptional, and the most common event is for the patient to sleep more or less tranquilly in the early part of the night, so far as the cough will let him, and to wake up in the early morning to find himself drenched with perspiration. These are the colliquative sweatings of older writers. Some night-sweating is not uncommon even in early stages.

Loss of Flesh and Strength.—Emaciation is the rule in phthisis; it may be one of the earliest symptoms, and may give a note of warning, when the cough has been thought to be a mere bronchial catarrh; towards the end of a chronic case the emaciation is extreme. If, as a result of treatment at any time, the patient improves, he commonly puts on a little flesh, or at any rate remains stationary. Exceptionally, nutrition is maintained fairly well, even when the physical signs show that there is a considerable and even apparently active lesion. The muscular power is soon enfeebled, and the patients lose energy, become languid and unfit for prolonged exertion, whether of mind or body. The mental condition in many patients, however, is one of great hope and confidence; even when helpless in bed they often fail to realise how ill they are, and look for complete recovery could they but once get rid of the cough.

Anæmia.—The loss of nutrition is represented also in the blood, and the patient is commonly pallid, both in the early and late stages. But this anæmia is often masked by two other conditions—one is the hectic fever, which leads to a flushing of the cheek and lips, especially in the evening; the other is the imperfect aëration of the blood causing cyanosis.

Imperfect Aëration of the Blood.—This shows itself by cyanosis of the face especially in acute cases involving a large area of lung, and in chronic cases in which the right side of the heart has become somewhat dilated. Another result of the bad aëration of the blood causing chronic retardation of its flow in the distal parts of the circulation, is the *clubbed fingers*, or *ungues adunci*. The finger loses its tapering form, and the last joint becomes thickened, especially from palmar to dorsal surfaces, but also transversely. The nail is unusually convex from base to tip, and seems to curve over the end of the finger. The appearance is more pronounced on account of the wasting of the rest of the finger. It is a very characteristic feature of phthisis, not indeed peculiar to it, but present in a large proportion of the cases. The same change may be seen in the toes.

Complications.—It will not be necessary to do much more than enumerate the various complications that occur in phthisis, as they are described in different parts of this volume under the particular organs concerned. Many of them result from the formation of tubercles in other parts of the body. They are more common in the long-standing chronic cases.

RESPIRATORY ORGANS.—Tubercular disease of the larynx (*see* p. 397) is common in phthisis, and a considerable proportion of chronic cases in the third stage are characterised by the husky cough and hoarse or whispering voice of laryngitis, which greatly aggravates the distress of the patient. In exceptional cases the symptoms of laryngeal mischief precede those of the lung lesions, but it is doubtful if the tubercle is ever actually deposited in the larynx before it is formed in the lung. Very rarely indeed does this complication either directly cause death or require tracheotomy.

Pleurisy.—This is so common as to be almost a part of phthisis. An old phthisical lung is usually adherent to the chest by a thick fibrous layer and pleuritic lymph forms nearly always over tubercular lesions that approach the surface. Sometimes fluid is poured out, and this is mostly a sero-fibrinous effusion, less often an empyema. The former may be absorbed, or cured by tapping.

Pneumothorax.—This occurs in a very small proportion of cases, but phthisis is by far the commonest cause of pneumothorax. If the liquid contents of a cavity escape into the pleura, an acute pleurisy may be set up, effusion takes place, and there will not only be a pneumothorax, but a pyo-pneumothorax, or less often a hydro-pneumothorax. Pneumothorax in these different forms may be quickly fatal, or it may slowly disappear, or even while persisting may not very seriously increase the respiratory difficulties of the patient. Much depends on the side affected. If the communication occurs on the side of the most extensive disease, the dyspnœa may not be much aggravated; if on the side of the healthier lung, the patient's only breathing surface may be at once rendered useless (*see* p. 486).

CIRCULATORY SYSTEM.—The heart wastes in chronic phthisis, but not to the same extent as in a fatal case of cancer. In some more chronic and fibroid forms the right ventricle is dilated and thickened to a moderate extent. The arterial erosions and small aneurysms of the pulmonary artery in cavities in the lungs have been already mentioned.

Femoral thrombosis is frequent in the last days of patients suffering from phthisis: it is more common on the left side.

ALIMENTARY SYSTEM.—*Parasitic stomatitis*, or thrush, occurs under the same circumstances as femoral thrombosis.

Anorexia, *indigestion*, *nausea*, and *vomiting* are common accompaniments of phthisis more or less during its whole course. A capricious appetite and a distaste for fat in every form have been noticed even before definite symptoms in the lungs. In the last stages sickness or loathing for food is so marked, that it is one of the chief difficulties of doctor and nurse to get the patient to take anything at all. The most extraordinary things are fancied by the patient at one moment, only to be rejected directly they are put before him.

Diarrhœa is common in late stages; it may be due to simply catarrhal conditions, to tubercular ulceration of the ileum, or to lardaceous disease. The stools are variable: sometimes yellow in colour, and containing a little mucus, or blood. Large hæmorrhages are not often seen.

Peritonitis is very rarely the result of perforation of a tubercular ulcer. More often it is due to tubercles in the peritoneum, but it is not a common complication.

Lardaceous disease of the liver, spleen, kidneys, and intestines occurs in numerous cases of phthisis—about 20 per cent. (Powell).

Fatty liver is somewhat more frequent.

Tubercular disease of the *epididymis* and *vesiculæ seminales* in men, and of the *uterus* and its *appendages* in women, occasionally occurs, but these lesions do not usually form prominent complications at the bedside. *Fistula in ano* is occasionally associated with phthisis.

NERVOUS SYSTEM.—Here also tubercle occurs, generally as tubercular meningitis, which is a cause of death in a small number of cases. This secondary tubercular meningitis is often very rapid; perhaps the earlier indications are lost in the general symptoms already present.

Pains are frequent: those about the chest are attributed to pleurisy; others occur in the limbs, and *peripheral neuritis* may affect the nerves of the extremities.

ADDISON'S DISEASE of the supra-renal capsules is sometimes associated with phthisis.

ACUTE NEPHRITIS, apart from tubercles or lardaceous disease, is an occasional complication.

GENERAL MILIARY TUBERCULOSIS is one of the fatal complications of phthisis. The lungs, liver, spleen, and kidneys are the organs commonly affected, and sometimes the cerebral and spinal meninges (*see* p. 137).

BONES AND JOINTS.—Tubercular disease of joints, caries of ribs with subcutaneous abscesses, caries of the spine with psoas abscess, and other similar lesions may coexist with the pulmonary disease.

Fatal Termination.—Death takes place in the following ways: —Exhaustion, hæmoptysis, pneumothorax, meningitis, perforative peritonitis, and uræmia.

Exhaustion accounts for the larger number of cases, and it is explained by the pyrexia, the loss by expectoration, sweating, and diarrhœa, and the deficient supply of nutriment from anorexia, nausea, and vomiting. Occasionally it comes on suddenly in a patient actually going about, and may then be mistaken clinically for the result of pneumothorax or of hæmorrhage.

Pneumonic Phthisis (*Scrofulous Pneumonia*).—This begins very much like an attack of acute pneumonia, with pain in one side, high fever, chills, and night-sweats, cough and expectoration. The physical signs also are those of pneumonia; but they are most marked at the apex, and spread downwards. Dulness, bronchial breathing, and bronchophony are accompanied by coarse mucous râles, consonating râles, and loud clicks. Often the condition is much more marked in one lung than in the other. The mischief extends rapidly: the pyrexia is severe, there are profuse sweats, appetite is entirely lost, and prostration becomes extreme. The indications that the lung is breaking down are more and more marked; the temperature assumes an intermittent type, the sputum is purulent, and contains *débris* of lung-tissue. The illness is often fatal in the course of from five to twelve weeks, either by exhaustion, or by hæmoptysis, which is always abundant if it occurs at all; or by pneumothorax from the opening of one of the rapidly formed cavities into the non-adherent pleura. The lungs are solid from combined hepatisation and caseation; and there are numerous small cavities in every part of the organ, with ragged irregular walls, and purulent contents. In this pneumonic and caseous material definite miliary tubercles cannot be found; but bacilli have been often seen in such lungs.

Occasionally the process may not be at once fatal; partial recovery takes place, and the patient lingers on for some years.

Fibroid Phthisis.—This form may supervene upon chronic pleurisy and chronic pneumonia, and occurs in association with the prolonged lung-irritation of certain trades. It is extremely chronic, and often affects one lung only. Clinically, the case is distinguished by the evidences of contraction of the diseased lung: the chest is sunken, the heart is displaced to the affected side, the opposite lung may extend its resonant area in the same direction; the spleen and stomach on the left side, or the liver on the right side, may be drawn far up into the chest. The physical signs of cavities are chiefly at the apex, as in other cases of phthisis; but impaired resonance, bronchial breathing, and bronchophony are perhaps present over the whole of the affected lung. If the other side is involved, it is only at the apex. The symptoms are cough, purulent expectoration, and dyspnœa; the cough is frequently hard and prolonged, and the sputum may be fœtid from retention. There is, as a rule, no constitutional disturbance; there is no sweating, and the temperature is normal. After some time the right ventricle of the heart becomes dilated, and cyanosis and dropsy ensue. Lardaceous disease may result from the continued profuse discharge, and diarrhœa and albuminuria assist the fatal termination. Hæmoptysis occurs, but is not constant. *Post mortem*, the lung is found contracted to one-third or one-quarter its normal size, firmly adherent to the chest by a dense thick fibrous layer, and presenting a quantity of dense white or grey fibrous tissue, which contains caseous or cretaceous deposits, vomicæ, and dilated bronchial tubes. Tubercular or fibroid change may be present to a smaller extent in the opposite lung.

Diagnosis of Phthisis.—In advanced cases the physical signs of consolidation or cavity at the apex of one lung, with purulent or sanguineous sputum, fever, and emaciation, determine the diagnosis, especially if the fever is intermittent or decidedly remittent, with night-sweats, and if the illness is of some duration—that is, longer than a pneumonia. The detection of tubercle-bacilli in the sputum is conclusive, and the same may be said of lung-tissue, where gangrene of the lung is not in question.

It is much more difficult to be certain of the presence of phthisis in its early stages. Cough, expectoration, wasting, and even hæmoptysis often precede the physical signs, and repeated examinations may be necessary to determine the point absolutely. The most conclusive physical signs are impaired resonance with deficient entry of air at one apex, and deficient entry of air with râles on inspiration.

If hæmoptysis be the chief symptom, it must be clearly made out that the blood really comes from the lung, and not from the stomach. The descriptions of a patient are often very unsatisfactory or misleading. Blood from the lungs should be coughed up, and should be bright red and frothy; it is often preceded by a tickling at the throat, not by a sense of nausea. *Anæmic* girls often speak of finding blood on their pillows on waking. This is, no doubt, from the gums or cheek, and is generally diluted with saliva. *Malingerers* may produce blood in a similar way. In *purpura*, blood may be expectorated actually from the lung, but the cause will be readily distinguished by the associated symptoms. Hæmoptysis is frequent in *heart disease;* especially early mitral stenosis in young people may give rise to cough, hæmoptysis, and shortness of breath; and the heart should therefore always be examined in a doubtful case.

Phthisis is sometimes masked by *bronchitis.* The accentuation of the physical signs at one or other apex should help one, as well as the history of the illness, hæmoptysis if present, and the detection of bacilli in the sputum. Phthisis may be sometimes simulated by *empyema,* which will produce fever, sweating, emaciation, cough, and purulent sputa; but examination will show the predominance of physical signs at the base in empyema, while the apex is clear. The two may, of course, coexist, and in any case exploration with a needle and syringe will settle the point. It is well to remember, also, that *pleuritic effusion* sometimes causes a tympanitic note under the clavicle, with bronchial breathing and bronchophony, which may for a moment make one think of phthisis.

Prognosis.—Complete recovery of the *lung* from tubercle perhaps never occurs; it must always leave a scar of fibrous tissue or some calcareous deposit, and destroy a small portion of lung-tissue. But the *patient* may, and sometimes does, recover. This is chiefly in cases that are placed under the most favourable hygienic and climatic conditions on the first appearance of symptoms; whereas cases which reach the second and third stages, or which cannot, from the pecuniary or other circumstances of the patient, be thoroughly dealt with, do, as a rule, go on to a fatal result. This may occur in a few months or the case may last three or four, or even ten or fifteen, years. In such cases, the illness is not uniformly progressive, but is interrupted by long intervals of marked improvement. Much fever, free hæmoptysis, abundant expectoration, and physical signs indicating rapid changes in the lung, which can best be estimated from two or more examinations at a few weeks' interval, are unfavourable. But in cases of the ordinary type it is very difficult to form a safe opinion as to how long the fatal end may be postponed, and the young physician is especially warned against making rash predictions as to when his patient will be dead.

Treatment.—Attempts have been made to cure phthisis by agents which will destroy the bacillus. Koch's *tuberculin* * was such an attempt, and more recently creosote, guaiacol, and guaiacol carbonate have been largely used, and are believed to have done much good in some cases. They may be given in increasing doses up to large amounts. From 10 to 100 minims of creosote in capsules or dissolved in cod-liver oil, 10 to 60 minims of guaiacol in capsule, or 20 to 90 grains of guaiacol carbonate in cachet or wafer, can be given daily. But our chief reliance must be on the improvement of the body and its tissues in every possible way, so that it may be enabled to resist the inroads of the disease, or rather that the tissues may become less fitted as a soil for the bacillus, and less readily excited to inflammation in its different forms. This indication is met by providing for the individual perfect hygienic surroundings, pure air, good and abundant food, and sufficient exercise. In early stages, when the disease is not very active or advanced, a change of climate is most desirable, the object being to obtain a pure and bracing air, which the patient may enjoy for several hours daily outside the house, without the risk of catching cold, and without undue strain to his lungs. This is best obtained by a sea voyage to Australia, New Zealand, or the Cape, or by residence at the Cape, or New Zealand, or at high altitudes in Switzerland (*e.g.*, Davos, Wiesen, or Moloja), or with less success on the English south coast—at Hastings, Dover, or Folkestone. At such places the patient may spend the whole of the winter, thus avoiding the cold, damp, and fog of that season in the greater part of England, and he may return to his home in the summer, when the weather is more tolerable. With the advent of winter he should again seek the climate that he has found suitable to him. Such a proceeding is, of course, not practicable to thousands of patients, but no other method of treatment at present known can really take its place. In advanced stages, however, in markedly hæmorrhagic cases, and in cases complicated by rheumatic tendency, feeble circulation, bronchitis, emphysema, or albuminuria, the bracing climates and high altitudes are likely to be injurious; and patients in the third stage are more often benefited by a warm and rather moist climate, combined with pure air, such as is to be found at Bournemouth, Torquay, Penzance, and neighbouring places in England, and abroad at Mentone, Cannes, San Remo, and the Riviera generally. Other places south of England do good service in this way, such as Algiers, the Canaries, Madeira, and Egypt. It is scarcely necessary to say that the dwelling-rooms should be well ventilated, and that the patient should be warmly clad, with woollen materials next to the skin. In less favourable climates, but in specially constructed sanatoria, such as that of Falkenstein, the open-air treatment of phthisis has been conducted with some success; and it is to be hoped that institutions of the kind may be established in the British Isles.

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* This method of treatment was introduced, with much promise, in 1890, was forthwith tried over the whole civilised world, but was practically abandoned after a few months as being useless, or even dangerous. It consists in the subcutaneous injection repeatedly of small quantities of a glycerine extract of pure cultivations of tubercle-bacilli, containing a chemical substance produced by these organisms (*tuberculin*). After each injection febrile reaction with increased local changes takes place, lasting twelve to thirty-six hours. The dose is repeated till toleration is reached, and then it is increased: and this is repeated until the dose reaches, perhaps, fifty times the first dose, when the process is stopped. The local changes were supposed to check the development of the bacilli. If the person is in health no reaction occurs: a single injection was therefore proposed for diagnostic purposes. This is used to detect tuberculosis in cattle now, as *mallein* is used for glanders in horses (*see* p. 155).

The diet should be plain, nutritious, and abundant; stimulants may be taken in moderation, but are not vitally necessary in the early stage, and certainly should not be allowed to interfere with digestion. Nutrition may be furthered by cod-liver oil, taken in doses of 2 to 4 drachms two or three times a day after meals, and by quinine and iron in small doses so long as the digestion is perfect. Any defective action on the part of the stomach must be at once met by suitable remedies (*e.g.*, alkalies and nux vomica), and the importance of good gastric digestion as an aid to nutrition must never be lost sight of in the treatment of the various symptoms and complications.

No very satisfactory results have been derived from the ***surgical*** **treatment** of phthisical cavities.

Symptomatic Treatment.—*Cough.*—This, of course, depends on the progress of the disease, and will lessen with the improvement brought about by general treatment. If it is painful, frequent, or keeps the patient from sleep, it may be treated by small doses of opium or morphia, in combination with expectorants; for instance, tr. camph. co. with tr. scillæ, or liq. morphinæ hydrochlor. with vin. ipec., or syr. papav. with camphor water, or a few minims of chlorodyne. Counter irritation by tincture of iodine, or by small blisters is also useful. A morning cough, which gets rid of accumulated secretion, may be usefully promoted by a little ammonium carbonate.

Night Sweating.—This can be generally checked by 1 minim of liq. atropinæ sulph. given in a little water at night; or 2 or 3 grains of oxide of zinc in a pill, with or without $\frac{1}{6}$ grain of extract of belladonna. Arseniate of iron ($\frac{1}{5}$ grain), or picrotoxine ($\frac{1}{50}$ grain), or tincture of nux vomica may also be used.

Hæmoptysis.—The patient should be kept in bed in the semi-recumbent posture, an ice-bag should be placed on the front of the chest over the lung from which the blood is supposed to come, the diet should be fluid, cold, and given in small quantities at a time, and one of the following styptics should be ordered:—Opium, with dilute sulphuric acid; pil. plumbi cum opio; digitalis, opium, and quinine—a grain of each; gallic acid; tr. hamamelidis; or liquid extract of ergot. If the bleeding is at all profuse, 3 grains of ergotin should be injected subcutaneously or into muscle.

Diarrhœa.—For this, one must carefully regulate the diet, and use the vegetable astringents, mineral acids, opium, sulphate of copper in $\frac{1}{4}$-grain doses, or subnitrate of bismuth.

Laryngeal Ulceration.—(*See* Tubercle of the Larynx, p. 397.)

Pleuritic pains are frequent, and are often relieved by painting the surface with tincture of iodine. Anodynes internally may be necessary. Pleuritic effusion delays the progress of the disease in the corresponding lung, and should not be tapped unless its pressure is extreme. An empyema requiring evacuation may be aspirated.

Pneumothorax.—For the acute pain, on its first occurrence, a morphine injection may be required. Later on, the complication may cause but little trouble and may be left alone. If air accumulates so as to give serious trouble from distension, it should be let out by a trocar and cannula.

Excessive Expectoration.—The inhalation of antiseptics by means of respirators is a useful means of treating this symptom. In the apparatus is placed a piece of lint saturated with some volatile antiseptic, such as eucalyptol, carbolic acid, or thymol, or a mixture of these. The patient wears it over his mouth for one, two, or three hours at a time, and breathes the vapour into his lungs. As a consequence, the expectoration often becomes less abundant, and loses any offensive characters it may have. Under these circumstances, patients have gained weight, and improved a good deal, but the method cannot be supposed to destroy the tubercle-bacillus itself.

Prevention.—The rules of life which are recommended for a patient with early phthisis apply to children born of phthisical parents, at least as long as they can be enforced; it is by such means that they can best unfit their tissues for the reception of the bacillus. Phthisical persons about to marry should be informed of the risk that their offspring may develop the disease;

a risk which is thought to extend to non-phthisical unions, if both husband and wife come of phthisical stock. One must not forget the possible conveyance of the disease from person to person under some circumstances, especially its indirect transmission in the manner suggested (p. 454) between relatives sleeping together, or brought into constant close contact, as when one dying of phthisis is assiduously nursed by another. The phthisical should not sleep in the same bed with a healthy person. Underclothes and bed-linen should be scalded before being washed. The sputa should in all cases be ejected into antiseptic fluid (5 per cent. carbolic acid solution); and they should finally be rendered innocuous by exposure to boiling water for ten minutes. Tuberculous mothers should not suckle their infants.

PULMONARY ASPERGILLOSIS.

The lungs are affected with local caseating or suppurative lesions as the result of glanders (p. 155), of actinomycosis (p. 162), and of a fungus, the *Aspergillus fumigatus*, of the order Ascomycetes. This rare disease occurs amongst pigeon-feeders, in consequence of the fungus being contained in the seed, which the feeder puts into his mouth for transference to that of the pigeon. The lesions are like those of tubercle, with cavities, fibroid changes, or emphysema according to the rate of progress and the resistance of the tissues, and the symptoms are dyspnœa, cough, expectoration, and hæmoptysis. The fungus does not spread to other parts of the body, and recovery may take place spontaneously.

CANCER AND OTHER TUMOURS OF THE LUNG.

Cancer of the lung may be either primary or secondary. The latter is more common, and occurs mostly in the form of nodules scattered irregularly through the substance of the organ, or forming plates covering the surface of the pleura. The original seat of the growth may have been in the breast, the liver, or stomach, or one of the limbs. A true primary cancer of the parenchyma of the lung is quite rare, and the disease that has gone by this name has generally spread from the bronchial glands or the peri-bronchial tissue. It commonly invades the lung at the root, and may spread thence into the pulmonary substance, chiefly following the course of the branching bronchi. The organ may thus be largely converted into a mass of new growth. But other changes take place simultaneously; the cancer may grow into the lumen of the bronchial tubes; here and there the growth breaks down into a granular detritus; and adjacent portions of the lung may become pneumonic or gangrenous. The cancer is more often of the softer or medullary variety.

Symptoms.—These vary with the position and distribution of the cancer growths—that is, according as they are seated in the main bronchus or bronchi, or are disseminated throughout the lung, or form one continuous mass or infiltration, involving a large portion of the organ.

1. Cancer involving the bronchus usually causes obstruction, the symptoms of which have already been described. Sometimes primary cancer of the mucous membrane causes death by profuse hæmorrhage.

2. When the lung is the seat of numerous nodules of growth, scattered indiscriminately through it, the patient suffers at first, at any rate, but little discomfort, and the physical signs are not very distinctive. Percussion resonance is normal, and the only change that may be observed is a diminution of the respiratory murmur all over the chest. In other cases the cancer nodules are more numerous, or set up bronchitic changes in the neighbouring bronchi; and more decided, though scarcely characteristic symptoms, may be produced. These are dyspnœa, very rapid breathing, lividity, frequent cough, and mucous expectoration; and on auscultation numerous rhonchi and râles are heard over the whole chest. The condition bears some resemblance to miliary tuberculosis, but the temperature is generally normal.

3. Uniform infiltration of the lung is insidious in its course, and produces cough, dyspnœa, and expectoration of mucus, which is sometimes tinged with blood, and sometimes mixed with larger quantities; occasionally it has a dark colour, and resembles currant jelly; but hæmoptysis is not very common. Pain is not generally a prominent symptom. The physical signs are those which must result from the infiltration of the lung with a solid material, at the same time that the bronchial tubes are filled up or blocked by the new growth. There is dulness, with absence or deficiency of breath-sounds, of vocal resonance, and of tactile vocal fremitus. There is thus a general resemblance to pleural effusion, which is often very deceptive. If the growth is considerable, the resemblance is increased by expansion of the chest-wall and displacement of the mediastinum; but a simple infiltration of the lung without large masses may be accompanied with contraction of the side, and resembles rather cirrhosis or chronic pneumonia, or phthisis, or chronic pleurisy with partial absorption of fluid, and retraction of the side. Exceptionally, from the breaking down of the cancerous material, cavities are formed which produce physical signs like those arising from tubercle. Sometimes the growth is accompanied by pleuritic effusion, and a serous fluid is poured out which is very

apt to contain blood from rupture of the vessels of the new growth. The bronchial, cervical, and axillary glands become enlarged; and extension to the mediastinum may lead to symptoms of pressure, such as œdema of the head, neck, and upper extremities, abductor paralysis of the vocal cords from pressure on the recurrent laryngeal nerves, obstruction of the trachea or one bronchus, or dysphagia from pressure on the œsophagus.

Cancer of the lungs, no less than that of other organs, is accompanied by progressive emaciation and loss of strength, and ultimately, in the course of from six to twelve months, death takes place, generally from exhaustion.

Diagnosis.—In case of malignant disease of other organs, the presence of unaccountable dyspnœa should make one think of cancer of the lung; and in cases where the pulmonary symptoms are most prominent, the presence of large hard glands in the neck, or a tumour of the testis, or a rigid spine from implication of the vertebræ, may sometimes give the required clue. Extensive infiltration of the lung is most easily confounded with *pleuritic effusion;* and in elderly persons with the symptoms and physical signs of fluid, the possibility of cancer should not be forgotten. Exploration with the needle, or a trocar and cannula, or aspirator may be necessary, and particles may then perhaps be obtained for microscopic examination. The sputum sometimes provides similar evidence. If in an elderly person a pleural effusion returns again and again one may suspect cancer, and all the more if the serum contains much blood; but a simple pleurisy often gives blood-stained fluid, from the rupture of vessels newly formed in the lymph or adhesions.

The **Prognosis** is bad, and the **Treatment** must be confined to relieving pain and cough, procuring sleep, and supporting with good nourishing food. A liquid effusion accompanying the cancer may be aspirated, but it will probably return quickly.

Other forms of tumour occur in the lung, and are, as a rule, secondary to similar tumours elsewhere. Such are sarcoma, osteosarcoma, and enchondroma. Epithelioma of the œsophagus sometimes invades the lung directly.

HYDATID OF THE LUNG.

This parasite affects the lung in two ways. First, a cyst may form in the lung apart from, or even without, its occurrence in any other region; secondly, the lung may be invaded by a cyst in an adjacent organ rupturing through the parts which separate them. This is most common in hydatid of the liver.

A *primary hydatid of the lung* is very rare. It forms a globular cyst, with all the characteristics of the parasite as seen in the liver (*see* Hydatid of the Liver), but it is not generally surrounded by such a dense connective-tissue cyst. It is rather more frequent at the base than at the apex. It may be the only cyst in the body, or there are others at the same time in the liver, spleen, brain, or elsewhere.

Symptoms.—These depend upon its size, and upon the change it sets up in the surrounding lung. It may be so small as to yield no symptoms whatever. If larger, it must compress the lung-tissue, and it gives rise not uncommonly to hæmorrhages, and may cause pneumonia, or even gangrene. The patient has cough, dyspnœa, pain, and sometimes hæmoptysis. If the cyst ruptures, secondary cysts may be expectorated, and will at once reveal the nature of the case. The physical signs, if any, are dulness, with more or less deficiency of vesicular murmur. If the cyst ruptures and the contents are discharged by the bronchus, the distinctive signs of cavity may be heard.

The lung may be *secondarily* invaded by *hydatids of the liver*. The pulmonary symptoms are then preceded by signs of hepatic disease, such as pain and tension in the hepatic region, some enlargement of the liver, and perhaps jaundice; and the earliest change in the lung is compression of its lower part by the enlarging cyst. Increasing pain and distress, with more or less collapse or prostration, may mark the implication of the lung itself; and soon cough of paroxysmal nature is followed by the expectoration of bile-stained hydatid skins, or small and perfect cysts. More or less pneumonia, or even gangrene of the lung, may result, and thus the case may end fatally. But it is not uncommon for the inflammation of the lung to be limited in extent, and the whole of the hydatid may in time be expectorated through the bronchi, and the patient thus recover completely.

The **Diagnosis** of primary hydatid is generally obscure, and the case is most likely to be mistaken for phthisis, especially if the cyst is situated at the apex. A girl with cerebral tumour in Guy's Hospital had hæmoptysis, and was thought to have tubercle of the brain and pulmonary phthisis; but a hydatid cyst was found in both brain and lung. *Secondary* hydatid is generally recognised by the preceding hepatic trouble, and the appearance of bile and hydatid skins in the sputa.

Treatment.—No internal treatment can kill the parasites in the lung. If the diagnosis of a cyst sufficiently near the surface could be made with confidence, it might be treated by the surgical methods applicable in hydatid of the liver. Hydatid of the liver opening into the lung is commonly beyond the reach of surgical interference, and the treatment must be symptomatic, and in the main supporting.

SYPHILIS OF THE LUNG.

Apart from the ulcerations of the bronchi, with resulting stenosis, which have been shown to be due to syphilis, the lung-tissue itself may exhibit the effects of the disease in two forms certainly. One is that of the ordinary *gumma*, which is extremely rare in adults, though more common in infants, and gives rise to no recognisable clinical symptoms. The other is the so-called *white pneumonia* of syphilitic infants. The lungs are enlarged, white, dense, and firm; their section is smooth and opaque; they are sometimes resistant, at others easily broken down. This condition may affect the whole lung, or one part may be uniformly altered, while the other contains only isolated areas. Ziegler describes it as a diffuse cellular inflammation of the lung, often accompanied by desquamation and fatty degeneration of the pulmonary epithelium. Wagner thought that it was entirely due to thickening of the alveolar wall, by which the cavity of the air-vesicle was gradually obliterated. As it is found chiefly in stillborn children it has but little clinical importance.

The extent to which syphilis may affect the lung in adults otherwise than by gumma has been the subject of much discussion. Destructive changes with bronchiectatic cavities may take place as a result of bronchial or tracheal stenosis; but it is not yet clearly shown that a "syphilitic phthisis" occurs in any other way. When a destructive disease of the lung occurs in a syphilitic subject, the possibility of its being tubercular should be fully considered.

DISEASES OF THE PLEURA.

PLEURISY.

Pleurisy, or inflammation of the pleural membrane, results in the effusion of lymph, or serous or purulent liquids.

Some of the causes of pleurisy are easily recognised, such for instance as injury by fractured ribs: and the extension to the pleural surface of (1) lesions in the lung, like those of pneumonia, phthisis, pyæmic abscesses, cancer, tubercle, or hæmorrhagic infarcts; (2) lesions of the parieties, such as abscesses in the axilla, breast, neck, or abdominal cavity. But a much larger number of cases are apparently spontaneous, or are preceded by exposure to cold; of these it has now been shown that a very large proportion then or subsequently show tubercular connections. Either they have a history of tubercle, or they afterwards die of phthisis or other tuberculous lesions. But it results from other infective agents, such as those of scarlatina, measles, rheumatic fever, and pyæmia, and it is a frequent complication of Bright's disease.

The micro-organisms found in cases of pleurisy are the following: Pneumococcus, streptococcus, staphylococcus, and tubercle-bacillus. They are often combined, as, for instance, pneumococcus or tubercle-bacillus, with streptococci or staphylococci; the last are not commonly found alone. In the sero-fibrinous effusions of tubercular pleurisies, tubercle-bacilli are often absent. In purulent effusions of children, pneumococci are mostly found, and in those of adults streptococci are more common. Tubercle-bacilli may be present in tubercular purulent effusions.

Morbid Anatomy.—The first stage of pleurisy consists of dilatation of the vessels of the pleura, quickly followed by exudation of the white corpuscles and fibrin on the free surface. Thus the membrane is at first minutely injected, but in the earliest visible stage, the naturally shining surface is rendered dull by the fibrin, which can be detached as an extremely delicate membrane. If the exuded material is more abundant, it forms thicker layers, firm or pasty, generally rough on the surface, or villous, or reticular. Pleurisy may go no further than the formation of fibrin on the surface, and is then called "dry"; more often, the fibrin is soon followed by the exudation of a serous or sero-fibrinous fluid, which may accumulate to the extent of two or three pints or more in the pleural cavity. This fluid has a yellow or greenish-yellow colour, a specific gravity of 1005 to 1030, often 1015 to 1018, and it becomes almost solid on boiling, from the albumen it contains. Not infrequently there are a few flakes of fibrin, or a quantity is deposited from the liquid a short time after its removal. The liquid is quite clear, or it is opalescent or turbid from the presence of corpuscles. In other cases the corpuscles are in sufficient quantity to form a thick layer at the bottom of the fluid that is withdrawn, and there is every gradation between this and the formation of real pus. Sometimes the liquid is more or less tinged with blood. Such blood may proceed from new-formed vessels in the false membranes, and is not necessarily an indication of malignant disease.

This effusion of fluid is one of the most important results of pleurisy. Confined within the cavity of the pleura, it must displace the lung from its relations to the diaphragm and the wall of the chest, and in proportion as more fluid is effused, the lung becomes collapsed. This is not at first due to the actual pressure of the fluid, but to the elasticity of the lung, which naturally favours its retraction; and, indeed, it may be found that even a considerable quantity of fluid in the chest may fail to escape, or escape but slowly, on puncture, being held in, as it were, by the natural retraction of the lung towards the mediastinum. But with a larger quantity a point is reached beyond the elastic collapse of the lung, and then this organ and the surrounding parts are actually subject to the positive pressure of the fluid, and the most extreme changes of position take place. An important difference in the effects upon the lung is that the larger quantities of liquid compress the bronchial tubes, whereas with less amounts only the air-vesicles are deprived of air; and this explains some differences in the physical signs. The pressure which forces the lung towards the mediastinum, pushes the mediastinum itself, with the heart and great vessels, towards the opposite side, bulges the wall of the thorax outwards, distends the intercostal spaces, and forces downwards the diaphragm with the subjacent liver or spleen.

In a large number of cases pleurisy recovers by absorption of the effused products. The liquid disappears in the course of days or weeks, and the lung and the chest-wall finally come into contact either by expansion of the former, or by a gradual sinking in of the latter, or by a combination of both processes. The layers of fibrin covering the two surfaces have already probably become partly organised by the growth of new vessels from the pleura, and the formation of fibrous tissue; and, uniting together, they form in time a permanent layer of *adhesion* between lung and chest-wall.

A purulent pleurisy, usually known as *empyema*, appears sometimes to arise out of a serous pleurisy, or *simple effusion*; but it is often found soon after the onset of the disease, and must then be primary. The early occurrence of empyema is most common in association with acute lobar pneumonia, in pyæmic and septic cases, after scarlatina, and as a result of perforation of the pleura from the lung or the abdomen. Its termination is by no means so favourable as that of simple effusion. Occasionally, no doubt, absorption takes place—that is, the fluid is taken up, the pus corpuscles become granular and fatty, and a caseous mass remains behind; or calcareous salts may be deposited in the residue. Sometimes an empyema finds its way through the pleural sac, either perforating the lung, so that the pus is expectorated; or "pointing" in one of the intercostal spaces, often the fifth, and bursting spontaneously if not opened by the surgeon. In either case air may find its way into the pleural cavity, and give rise to *pyo-pneumothorax*. Rarely an empyema opens through or behind the diaphragm into the abdomen. But, if unrecognised or untreated, it may remain a long time without perforating, with incomplete absorption, rendering the patient cachectic, and preparing the way for lardaceous degeneration of the viscera.

Both in serous and purulent effusions, the cavity is occasionally divided into two or more separate spaces, by adhesions between the lung and the parietes. The fluid is then said to be *loculated*; and the condition is of importance when the case is treated surgically.

Symptoms.—The onset of pleurisy is characterised by a chill or rigor, with severe pain in the side, caused or aggravated by the act of breathing. In the apparently spontaneous cases, the pain is at first commonly at the side of the chest, over the lower ribs; but in pleurisy determined by other lesions—as, for instance, phthisis—it may be situated elsewhere. The pain is cutting or tearing, and is intensified not only by breathing, but by coughing, sneezing, and every kind of exertion. The patient generally lies on his back or on the healthy side. There is mostly some pyrexia, with the usual accompaniments—furred tongue, loss of appetite, and malaise.

Physical Signs.—On examining the chest some impairment of movement on the affected side and deficiency of vesicular murmur at the painful spot are observed; but the characteristic physical sign is the *pleuritic rub*, or friction sound, which arises by the movement upon one another of the two pleural surfaces, roughened by exudation (*see* p. 385). The sound will vary with the degree of friction, and this may be so great that it can be felt by the hand placed on the chest, as well as heard with the stethoscope; on the other hand, one may fail to hear the rub in this first stage, if the patient is prevented by the intense pain from making the inspiratory movement necessary to produce it.

When liquid is effused, the two pleural surfaces become separated, the friction sound disappears, the pain diminishes, and symptoms and physical signs occur which are the direct result of the presence of liquid and the compression or displacement of the various organs which it affects. The chief symptom is shortness of breath, especially on exertion, and this dyspnœa will be in proportion to the amount of liquid in the pleural cavity. It is often scarcely observed when the patient is still, but becomes manifest when he moves about, or even when he talks. He lies on his back, or on the affected side, to allow the greatest freedom to the healthy lung. He may be entirely free from cough, or may have slight cough without expectoration. Fever is commonly present, the temperature ranging from 100° to 102°, and with it there are malaise, weakness, loss of appetite, and quick pulse. As the fluid gravitates to the most dependent part of the chest, small quantities are usually detected at the base behind, where there is absolute dulness, while vesicular murmur, vocal resonance, and tactile vocal fremitus are much enfeebled, or entirely absent. With a considerable quantity of fluid the following physical signs are observed:—On inspection, the affected side of the chest is motionless, and may be obviously larger than the other; the intercostal spaces, instead of being slightly depressed below the level of the ribs, are filled up (or "obliterated"). The heart is displaced: with effusion on the right side, its impulse may be perceived beneath or outside the left nipple; with effusion on the left side, an impulse is often felt in the intercostal spaces to the right of the sternum, generally the third, fourth, and fifth, even as far as the right nipple, and in rare instances beyond it. The dulness in such cases is observed in front, in the axilla, and behind, and is continuous with dulness on the opposite side corresponding to the displaced heart. The liver or the spleen may be pushed down, according to the side affected; and the descent of the diaphragm on the left side leads to dulness at the upper part of that space between the left lobe of the liver and the spleen, which normally yields gastric resonance. When the liquid is sufficient only to reach about two-thirds the height of the chest, there may be heard the peculiar modification of the percussion note under the clavicle and above the level of dulness, which is known as *Skodaic resonance* (p. 382). At this spot there are bronchial breathing and bronchophony; and sometimes heavy percussion elicits a sound closely resembling the cracked-pot sound of phthisical cavity. Over the dull area there are diminution or absence of breath-sounds, of vocal resonance, and of tactile vibration. Of these, the last is very constant, but the breath-sounds, instead of being absent, are sometimes bronchial, especially at the upper part of the area of dulness; and this is to be explained by the bronchial tubes at this level being still patent, although the vesicular tissue is collapsed and the lung inactive. The conditions for the production of bronchial breathing are thus the same as in pneumonic consolidation. Generally, however, the bronchial breathing is less loud and ringing—softer, or more distant, and often of high pitch or "whiffing." Occasionally, especially in children, the bronchial breathing is heard over the whole area of dulness. Where the breath-sounds are bronchial, the vocal resonance is often increased. It is especially in pleuritic effusion that the modification known as *ægophony* is heard (p. 386).

In extreme cases the whole of one chest is dull, back, front, and side, and from apex to base. Generally, bronchial breathing is entirely suppressed, except along the spine at the root of the lung, and ægophony is less frequently present than in more moderate degrees of effusion. The mediastinum and heart are displaced, and the diaphragm is depressed on the same side. The disturbance of the functions of the thorax may at length be fatal. Sometimes this takes place by sudden syncope, which has been explained by the displacement of the mediastinum bending and obstructing the inferior vena cava to such a degree, that a very slight additional disturbance may completely stop the supply of blood to the right auricle. In other cases death arises more gradually from the interference with the respiratory functions. The patient becomes more and more livid, rhonchi and mucous râles are heard in the hitherto healthy lung, and asphyxia finally ensues.

Very rarely the pulsation of the heart is communicated to a pleural effusion, either as a shock or wave transmitted to a large serous collection, or as a more localised, perhaps visible, pulsation in an empyema, pointing through the chest-wall.

Progress to Recovery.—Where recovery takes place spontaneously, the liquid disappears, sometimes gradually, at others with remarkable quickness. The upper parts of the area of dulness become resonant, the vesicular murmur returns, and often with it the friction sound is again heard, louder, longer, and over a much more extensive surface than was the case previous to the effusion. In addition, the rub is more distinctly palpable, but much less painful. While the liquid is being absorbed, and the lung is beginning to expand, the chest often falls in—a change which is first observed behind, where the naturally round chest becomes flat as compared with the other side. In extreme cases the chest is flattened in front and behind, these two surfaces meeting in a rounded angle at the axilla; at the same time, the dorsal spine is curved, with the concavity towards the affected side, the shoulder is depressed, and the angle of the scapula projects from the ribs. Frequently the physical signs at the base remain abnormal for some time, suggesting the continuance of liquid, but undoubtedly due to incomplete expansion of the extreme base, with or without a layer of new fibrous tissue (thickened pleura), the result of the inflammation.

In some cases a chronic pleural effusion results, the liquid neither increasing to a fatal result nor becoming absorbed.

Diagnosis.—In the early stages the pain of pleurisy has to be distinguished from other pains in the chest, the most common of which is *pleurodynia*, or muscular rheumatism. This is increased by movement, but is unaccompanied by fever or by rub. Intercostal *neuralgia* is distinguished by its relation to the nerves, and the tender points characteristic of neuralgia. It must be remembered that inspiration compresses the structures below the diaphragm, so that affections of the liver, spleen, or colon may give rise to pains aggravated by breathing.

In the second stage we have to consider, first, whether there is liquid in the pleural cavity; and secondly, what is the nature of the liquid, whether serum or pus. In acute cases, pleurisy and *pneumonia* are most likely to be confounded together; and it must especially be remembered that bronchial breathing is not in itself distinctive of the latter. Pleurisy is generally characterised by the absence of tactile vibration and the more absolute percussion dulness; and the larger effusions cause displacements of the heart, and of the diaphragm, which are conclusive. Generally, also, pleurisy is wanting in the very high fever, pungent heat of skin and flushed face of pneumonia; but should the sputum be rusty, pneumonia is certainly present. It must be remembered, however, that the two may occur together. The early signs of pleurisy are sometimes so little marked, and the effusion of liquid so insidious, that the local features of the illness may be for some time overlooked, and the patient may be thought to be suffering from *typhoid* or other form of fever.

In chronic cases, fluid may be simulated by most *consolidations* of the lung-tissue, whether from deposit in its substance or from external compression; such are some cases of tubercular consolidation, the induration which results from heart disease, cancerous growth in the lung, compression from the front by pericardial effusion, and from below by abscess, cancer or hydatid of the liver, cancer or hydatid of the spleen, or abscesses about the spleen and kidneys. The physical signs common to these conditions are dulness, loss of breath-sound, of vocal resonance, and of tactile vibration, simply because they compress or push up the lung in the same way as liquid effusion does, and do away with that spongy tissue in communication with the trachea and larynx, upon which the normal physical signs depend; but with less degrees of compression the breath-sounds are here also sometimes bronchial. Most of these changes affect the base of the lung, and no help can be gained from the absence of the signs (enlargement of chest or displacement of heart) which occur in the more abundant effusions. Cancer of the lung is especially deceptive because it may form a tumour, occupying and enlarging one side of the chest, and pushing the mediastinum and heart to the other side. It is more common in those over middle age, and diagnosis may be impossible without the aid of an exploration. In tumours, abscesses, or cysts rising from the abdomen, the upper limit of dulness may form a curved line convex upwards, instead of the horizontal line common in pleural effusions.

When pus is present there are not infrequently some differences which enable the fact to be recognised; but this is by no means always the case. Though empyema is often a sequel to simple effusion, the duration of symptoms is not a safe guide in its diagnosis. For, on the one hand, pus may be found within a few days of the onset, and, on the other, the liquid may continue serous for many weeks. Œdema of the chest-wall happens more frequently with pus than with simple serum. A better transmission of the voice by serum than by pus has been advocated by Baccelli, as a means of diagnosis between them, but it cannot be relied on. Slight flattening of the chest, showing that some absorption is taking place, suggests, but is not conclusive in favour of, serum. Effusions are especially likely to become purulent, when they have followed the eruptive fevers, such as scarlatina; and the early appearance of pus occurs in connection with acute pneumonia and with phthisis.

Perhaps the most important indications lie in the constitutional conditions which result from pus in the chest, as well as elsewhere. Thus a sallow appearance or even marked anæmia may be present; the temperature has often a hectic type, ranging from 98° or 99° in the morning to 102°, 103°, or 104° in the evening, and with this rigors or profuse sweatings may occur. But it must not be forgotten that any pleurisy may be accompanied by high fever and sometimes free sweating, and, conversely, the chest may be full of pus when the temperature is quite normal.

The sudden development of purulent expectoration, especially if offensive, in the course of pleurisy, is an important indication of empyema; and in cases of long duration the ends of the fingers tend to become thickened, or "clubbed."

The uncertainty attending these signs may at any time be removed by inserting into the chest a suitable exploring-needle connected with a glass syringe, into which the liquid may be drawn.

Serum may be present in the pleural cavity from other causes than pleurisy—namely, local and general dropsy. The physical signs may be the same, but *hydrothorax*, as the condition is called, generally follows upon disease of the heart, or Bright's disease, or pressure upon vessels by cancer in the chest; and there is an absence of the febrile accompaniments of pleurisy.

Prognosis.—Most cases of pleurisy without effusion, or with a sero-fibrinous effusion, get well, either under medicinal treatment or after surgical evacuation of the liquid. But the uncertainty of their subsequent history has been indicated. Empyema is more amenable to treatment in children than in adults, and more promising the earlier the pus is evacuated. The better results in children probably depend on the greater frequency in them of pneumococcal as compared with streptococcal pleurisy.

Treatment.—In the treatment the first consideration is the pain in the side. This may be generally met by the application of linseed-meal poultices, and the administration of opium or morphia internally, or morphia subcutaneously. The use of blisters, of leeches, or of cupping over the painful spot, also generally gives relief. F. T. Roberts strongly advocates strapping the affected side, by which means the respiratory movements are restrained; the pain is thus allayed, and inflammatory action probably in some measure checked. The strapping should be applied in broad strips from the spine to the sternum, alternate strips passing obliquely upwards and obliquely downwards, till the whole side is covered. The patient should be at the same time kept at rest, and placed on a diet suitable to his febrile condition. If effusion takes place, anodynes will be less needful, and salines, such as the acetate and citrate of potassium or ammonium, should be given, partly as refrigerants, partly for their effect upon the excretions of the skin and kidney, the increase of which will favour absorption of the effused liquid. After a time iodide of potassium, squills, or other diuretics may be added, and absorption may be encouraged by the application of the tincture or solution of iodine over the affected side. But in most instances the actual removal of the liquid by puncture becomes desirable. There are three cases in which this should certainly be done: (1) When the effusion fills the whole of one chest; (2) when the liquid, though not so abundant, has been present two or more weeks, and in spite of treatment shows no indication of being absorbed; (3) when the liquid is purulent, whatever its quantity may be. Some difference of practice obtains as to puncture in the early period of an effusion, since it is quite possible that absorption may take place without it, and for a time at least the effusion may be regarded as giving rest to the inflamed surfaces by separating them. But there are many arguments on the other side. We have little, if any, means of knowing how long an effusion may last if we do not interfere; as a rule, the longer the liquid is present, the more difficult it is for the lung to expand, both from changes in its substance and from the deposit of fibrin on its surface; the febrile temperature often falls directly the fluid is removed, even though it be only serum; and in many cases relief is at once afforded and progress towards recovery is continuous. Thus even a moderate quantity of liquid should be removed after a fortnight, if it is not by that time distinctly diminishing.

The liquid is best evacuated by the aspirator (Potain's or Dieulafoy's), which should be perfectly clean and aseptic. The site chosen for puncture may be the seventh or eighth space in the posterior axillary line, but when there is not much liquid it may require a puncture in the ninth space, near the angles of the ribs. It has been often urged that it is not desirable to draw off the whole of the liquid, because some more will almost certainly be secreted, and because the removal of a small quantity is enough to relieve the pressure on the absorbent vessels, and so facilitate the absorption of the remainder; but greater immediate relief is given by letting out as much as possible, and the cases seem to me quite as successful.

When, during the process of aspiration, the greater part has been removed, the patient frequently begins to cough, probably as a result of the admission of air to the newly opened bronchial tubes. The appearance of blood in a fluid previously bloodless should, as a rule, be an indication to stop the operation, withdraw the needle, and close the wound. Resonance generally returns at the upper part of the chest at once, but, in spite of the withdrawal of three or four pints from the chest of a grown-up man, the physical signs may persist for some time at the base, although the subsidence of fever and the very marked relief to the breathing show that no great accumulation is present. Occasionally the liquid may collect again and require a second or even a third tapping.

If an exploration by the needle shows that the effusion is purulent (empyema), the most certain method of treatment is to make a free incision under an anæsthetic into the eighth or seventh space, with the usual antiseptic precautions, insert a large red india-rubber tube into the aperture, and allow the pus to drain into the antiseptic dressings. In some cases, in spite of free drainage and thorough antiseptic treatment, the cavity continues to secrete pus, and the wound does not close. If this goes on too long the supervention of lardaceous disease is to be feared, and further effort should be made by removing a portion (1½ to 2 inches) of a rib or ribs adjacent to the wound. This widens the aperture for drainage, and allows the bony thorax to fall in upon the imperfectly expanded lung; where the ribs are very close together, resection may sometimes be done with advantage at the first operation. An empyema will sometimes be cured by a single aspiration, and this appears likely to occur especially when the pus is due to pneumococci; but I believe more time is saved by early incision. During the surgical treatment of an empyema the patient should be supported in every way by good food, stimulants, tonic medicines, such as quinine, iron and cod-liver oil, and, if possible, by fresh, bracing air.

HYDROTHORAX.

This term is applied to the collection of fluid in the pleural cavity, not as a result of inflammation, but in consequence of heart disease, or Bright's disease, or interference with the circulation in the chest by cancer or tumour. It is, indeed, dropsy of the pleural cavity; and the liquid contains less albumen and less fibrinogen than are found in pleurisy. Its physical signs are similar to those of pleuritic effusion, but the rub is, of course, absent. Arising, as it often does, from a general or central cause, it is much more often bilateral than pleurisy is; but occasionally a very large one-sided effusion may be merely dropsy. It is also stated, as another point of distinction, that when the position of the body is changed from recumbent to erect, or *vice versâ*, the line between dulness and resonance (that is, the upper level of the liquid) is likewise altered, rising higher in front when the patient sits up; whereas this mobility of the liquid is scarcely, if at all, noticeable in pleuritic effusions, which are confined to one situation by the surrounding adhesions. The recognition of hydrothorax, however, generally depends on the history and the previous existence of the diseases which cause it.

Its **Treatment** is mostly of secondary importance, being involved in that of the cause. As the liquid is almost certain to recur if removed, paracentesis or aspiration should only be performed where a very large effusion, whether on one side alone, or divided between the two, is seriously impeding respiration.

HÆMOTHORAX.

When blood in quantity is effused into the pleural cavity, the condition is called *hæmothorax*. It commonly results from surgical injuries, or rupture of a thoracic aneurysm. The **Physical Signs** are those of liquid. The **Diagnosis** will depend, in the case of aneurysm, on the previous history, and on syncope and pallor indicating rapid loss of blood.

PNEUMOTHORAX.

Pathology.—The presence of air in the pleural cavity, which the above term implies, may be brought about by any wound in the side which passes through the whole thickness of the chest-wall, and it is often produced by a fractured rib puncturing both layers of the pleura, so as to let out air from the lung into the pleural cavity, while the skin remains intact. Much more often, pneumothorax is the result of disease, and especially of phthisis, from the rupture or sloughing of the pleura over a vomica; and less commonly an empyema makes its way through the pleura into the lung, and air escapes into the pleural sac, so as to form a pneumothorax in addition to the empyema (*pyo-pneumothorax*). Similarly, pneumothorax occurs in the surgical treatment of empyema by incision. Rarely in acute pneumonia the pleura ruptures, and air escapes; or a pyæmic abscess or gangrene may lead to a similar result. Air may also enter the pleura in consequence of spinal or mediastinal abscess, burrowing into the pleura; and ulcer or cancer of the stomach, or cancer of the œsophagus, may let in air from the alimentary canal.

A pneumothorax may be described as open, closed, or valvular, according to the condition of the opening which created it.

Open Pneumothorax.—When air enters the chest from an external wound, and the wound remains patent, the lung collapses by its own elasticity; and not only the lung of the wounded side, but also the opposite lung contracts somewhat, and draws with it the mediastinum, so that lateral displacement of the viscera takes place, just as it does in liquid effusion. The same happens if the pneumothorax results from rupture of a cavity in phthisis, supposing the aperture to remain patent, so as to keep the pleural sac in communication with the bronchial tubes. In both these cases the mean pressure of the air in the chest is equal to that of the atmosphere.

Closed Pneumothorax.—When the aperture is small it may be quickly closed by lymph; further extravasation is prevented, and the air may then be completely absorbed. This happens in cases of laceration of the pleura by fractured rib, and sometimes, or to a less complete extent, in pneumothorax from disease of the lung. It can be understood that the conditions are much more favourable to absorption in pneumothorax from injury than in that from phthisis. In closed pneumothorax, the pressure has been found to be negative, and the displacement tends to be less than in the first case.

Valvular Pneumothorax.—A third possibility is that a shred of pleural membrane or lymph hangs over the aperture so as to form a valve. The air is then drawn in to the pleural sac by inspiration, but is unable to escape during expiration; the mean pressure becomes positive—that is, it exceeds the pressure of the atmosphere, and the displacement of viscera and distension of the chest may be extreme; thus, the heart may be pushed far over to the opposite side, and the liver or spleen may be driven down by the flattening or inversion of the diaphragm. A valvular opening may, like others, become closed by adhesions.

The amount of collapse of the lung, and displacement of the viscera, is influenced in different cases by the previous condition of the lungs. This is a point of importance from the large proportion of cases (nine out of ten) occurring in phthisis. If the lung is extensively diseased, or in great part adherent, the collapse will be less than if the lung, for the most part healthy, has only a small amount of disease, with cavity, at the apex. The entrance of the air into the pleural cavity may be otherwise harmless, especially in traumatic cases; but a serous or purulent effusion is commonly the result of the entrance of micro-organisms, especially when phthisis or a pyæmic abscess is the antecedent. If the effusion is serous, it may be absorbed together with the air, but a pyo-pneumothorax commonly persists unless dealt with surgically. The air of a pneumothorax differs from that of the atmosphere in containing very little oxygen, a large quantity of carbonic acid, and excess of nitrogen. Different analyses have given in 100 parts—oxygen, from 2 to 5; carbonic acid, from 6 to 16; nitrogen, from 80 to 90.

Physical Signs.—Over the affected side there is marked hyper-resonance of tympanitic quality, changing, of course, to dulness at the lower part when liquid is present at the same time. It is stated that in rare cases of extreme distension as the result of a valvular aperture, the percussion note may become muffled or actually dull. The respiratory murmur is often entirely inaudible, or a faint amphoric breathing is present. When this kind of breathing is loud or well marked, it is probably due to the aperture being patent; but a fainter sound may occur even when adhesions have shut off the lung from the pleural cavity. Vocal resonance and tactile vibration are generally much diminished, but bronchophony or pectoriloquy may be present. Sounds of a metallic or tinkling character are sometimes heard when the patient breathes, or speaks, or coughs; these are probably due to an echo of the vibrations by the side of the thorax. One characteristic sound is due to the dropping of fluid from the upper part of the chest into the liquid below, the noise being reverberated with almost musical quality. The *bruit d'airain*, or *bell sound* (p. 387), can also be elicited in cases of pneumothorax.

If there is also liquid effusion, its gravitation to the lowest part of the chest under all circumstances is well shown. If the patient is recumbent, the posterior part of the chest is dull, the anterior part is tympanitic; if the patient now sits up, the lower part of the chest, back and front, becomes dull, while the upper part, back and front, is resonant. If *Hippocratic succussion* be employed, a splashing sound will be obtained (p. 387).

The **symptoms** of pneumothorax are very variable, depending largely upon the amount of antecedent disease. If it supervenes upon a lung extensively diseased, it may add but little to the distress already present; if it occurs in a lung for the most part, or entirely, sound, the symptoms will be pronounced; lastly, if, in a case of phthisis with extensive disease on one side, pneumothorax occurs on the other side, the result may be quickly fatal. The symptoms in the severe cases are sudden pain, with a sense of something giving way internally, then distress of breathing, with more or less collapse, small pulse, lividity, and sweating. The breathing is shallow and rapid; the chest is distended on the affected side, and the intercostal spaces are depressed on inspiration.

These troubles may be aggravated until death takes place, within a few hours, or two or three days; or the first severe symptoms may subside, and comparative ease may follow, but generally with rapid breathing and orthopnœa.

Diagnosis.—*Emphysema* may be for a moment confounded with pneumothorax, but it is always bilateral unless compensatory on one side to disease on the other. A very large *cavity* in phthisis may sometimes simulate a localised pneumothorax in its hyper-resonance, feeble vesicular murmur, and tinkling sounds; but *bruit d'airain* must be rare in vomica, and the flattening of the chest over a cavity will generally serve to distinguish it from a pneumothorax. On the other hand, in some cases pneumothorax may be overlooked from the absence of any special symptoms at the time of its occurrence. *Ruptured diaphragm*, with escape of the stomach into the thorax, may closely resemble pneumothorax, especially as they may both arise from the same injury—a contusion of the chest. Pneumothorax may also be simulated by an unusually high position of the stomach in the chest in consequence of contraction of the left lung, and by abscess beneath the diaphragm containing air (*subphrenic pneumothorax*).

Prognosis.—In many cases it is the final event of phthisis, and death takes place in a few days; but some patients live for weeks or months with hydro-pneumothorax. Complete recovery with absorption of the air may take place, as in cases due to injury, in those following strain in whooping-cough, or pneumonia, and very exceptionally even in phthisis.

Treatment.—This is, in the main, palliative. For the intense pain and distress accompanying the rupture, opium or a subcutaneous injection of morphia (⅓ to ½ grain) should be administered, and hot poultices and fomentations should be frequently applied. Stimulants, as wine, brandy, or ether, may also be required. In cases of extreme distension it may be desirable to perform paracentesis, a trocar and cannula being inserted between the ribs over the resonant area. The air escapes, and the pressure within the chest is reduced, but the relief is, as a rule, only temporary. If the communication with the lung becomes closed, the air will probably be absorbed, and serum may be removed by aspiration; or if pus is present, it may be treated as in the case of empyema.

DISEASES OF THE ORGANS OF CIRCULATION.

PHYSICAL EXAMINATION OF THE HEART AND VESSELS.

LIKE the lungs, the heart is accessible to examination by the eye, the hand, and the ear. It comes into close proximity with the chest-wall between the anterior margins of the lungs, over an area corresponding to the lower half of the sternum on the left of the middle line, and the inner portions of the fourth and fifth left costal cartilages and the spaces below them. The *impulse* of the heart can be determined by inspection and palpation; the *præcordial area*, or the part of the heart exposed between the lungs, can be made out by percussion, and the *heart-sounds* can be studied by auscultation.

INSPECTION.

In health, the heart can be commonly seen to beat in the fifth intercostal space from half an inch to one inch within a line drawn vertically down from the nipple; this is called the *impulse*, or *apex-beat*. The former is the better term, because it is by no means certain that the point of visible impulse is always the actual apex of the heart, though it is very near to it. The impulse is normally limited to an area of half an inch in diameter.

In disease, various changes take place in the position and the character of the impulse. It may be in the sixth or seventh intercostal space, or in the fourth or third; it may be in the nipple-line, outside it, or in the axilla; it may be much nearer to the sternum than usual. Sometimes the beat of the heart can only be seen to the right of the sternum; or the impulse may extend over two or three intercostal spaces from the fifth upwards; or, in addition to the impulse in the usual position, there may be one below the ensiform cartilage (lower part of the right ventricle), or in the second left intercostal space (top of the right ventricle). Sometimes no impulse can be seen at all, either from feebleness of beat, or because the heart is overlaid by lung. In character, the beat may be unusually forcible, or heaving, or quick, or irregular.

Inspection also shows bulging of the chest-wall in some cases of great enlargement of the heart.

Movements in the epigastrium are often produced by the heart's contractions. A slight *systolic retraction* is not uncommon in healthy persons: a more marked retraction occurs with hypertrophy of the heart. If the right ventricle is chiefly hypertrophied a *systolic impulse* may be produced, and one slightly later in time occurs from the impact of the aorta, whether aneurysmal or conducted by tumour, and by a pulsating liver.

PALPATION.

The examination of the heart with the hand confirms much that can be seen with the eye as to the position and character of the impulse. In addition, the hand sometimes appreciates vibrations which correspond to sounds that can be heard with the stethoscope (*see* Auscultation). Over the base of the heart, mostly in the second left intercostal space, the closure of the pulmonary valves, which forms part of the second sound, can be sometimes felt as a sharp, short click; and in certain cases of valvular disease a *thrill* can be felt over a limited area, where the stethoscope reveals an abnormal sound, *bruit*, or *murmur*. Such a thrill may be felt at nearly all the orifices of the heart, but the most frequent is one that is felt near the apex of the heart in mitral stenosis. Aneurysms, perforation of the septum, and pericardial lymph also give rise to palpable vibrations.

PERCUSSION.

While the greater part of the chest is resonant to percussion, from the presence of lung, there is a small area over the surface of the heart which is not resonant. This *præcordial dulness* does not correspond to the whole anterior surface of the heart, but to what is exposed between the vertical anterior edge of the right lung and the oblique anterior edge of the left; and not all of this, because the sternum is normally resonant even up to its left border. Its limits are as follows:—Above, the upper border of the fourth costal cartilage; below, the upper border of the sixth cartilage; internally, the left border of the sternum; and externally, a vertical line from half an inch to one inch within the nipple. Around this dulness above, and to the right and to the left, is an area of less dulness, the outer limit of which corresponds to the outline, and therefore maps out the actual size of the heart: this reaches above the upper border of the third rib, or of the third space, to the left nearly the nipple line, to the right the right border of the sternum, but the dulness is least marked over this bone. The former central area of dulness is often called ***superficial***, or ***absolute dulness***, the latter surrounding band is called ***deep*** or ***relative dulness***. The former is best brought out by light percussion, the latter by heavy percussion. The lower limit of the heart's dulness cannot be discriminated from that of the liver, and the outline is assumed to lie between the impulse, and the lowest point of the right border of the dulness.

From the relation of the heart to the lung in this position, it results that the cardiac dulness is affected by changes in either of these organs. It is increased by enlargement of the heart, and diminished if the heart becomes smaller. It is, on the other hand, diminished by enlargements of the lung, which cover the heart; and it is enlarged by a retraction of the lungs, especially of the left, which exposes it more. An important cause of its enlargement is distension of the pericardial sac with liquid. Exceptionally, the area may be resonant from the presence of air in this sac. The area of præcordial dulness is shifted upwards, downwards, or to either side by anything which displaces the heart in these directions.

AUSCULTATION.

By means of the stethoscope we hear over the cardiac region, the well-known sounds of the heart: the *first*, or *systolic*, and the *second*, or *diastolic*. It is generally believed that the first is due partly to muscular contraction, and partly to closure of the auriculo-ventricular valves; and that the second is due to closure of the sigmoid valves. The first sound is heard best near the apex of the heart, and the second is heard best at the base.

Modifications of the Sounds.—The heart-sounds may be accentuated, diminished, or reduplicated.

Accentuation arises from several causes, amongst others from retraction of the lung, so as to bring the heart closer to the chest-wall; and from increased tension in the aortic or pulmonic arterial system, whereby the valves are caused to close with unusual force. This last condition affects, of course, the second sound, and it may be determined whether the aortic or pulmonic system is at fault by examining successively on either side of the sternum in the second intercostal space. On the right side the aortic second sound can be heard more or less apart from the pulmonary; on the left side, the pulmonary apart from the aortic. Accentuation of the first sound results from excessive action of the heart, and is common also in mitral stenosis.

Diminution of the sounds results from feeble action of the heart, from its being unusually covered by lung, as in emphysema, or from its being surrounded by pericardial effusion.

Reduplication may affect either sound. It is attributed to a want of synchronism in the closure of the two auriculo-ventricular valves, or of the two sigmoid valves; but the explanation is not satisfactory, because asynchronism of valvular closure seems to require asynchronism of ventricular action, a possibility not admitted by physiologists. As a fact, reduplication of the first sound is common in conditions of high arterial tension, such as gout and renal disease. Reduplication of the second sound is frequent in mitral stenosis.

Murmurs.—These are adventitious sounds, which accompany or replace those which are physiological. Originally described as *bruits de souffle*, they are now commonly called *bruits* or *murmurs*, and they are due mainly to two causes. The first is, that any narrowing or *obstruction* of a cardiac orifice, such as is produced by vegetations on the valves, or by union of the valves together, will produce vibrations in the current of blood forced through them. The second is, that if the valve does not perfectly close the orifice, some blood will flow back, or *regurgitate* into the cavity whence it came; and this leaking of a small stream through a narrow orifice or chink will be accompanied by vibrations which, if of sufficient amplitude, are audible as sound. Such sounds are generally explained by the theory of the *veine fluide*. In either case, the blood passes through a constriction, or narrow orifice, into a wider space beyond; this determines the production of a jet, or "fluid vein," which breaks up in such a manner as to produce vibrations among its own particles. In the same way may be explained a murmur which is sometimes heard as the result of the passage of blood through a perforation in the septum between the ventricles. In some cases the conditions requisite for the production of a *veine fluide* do not seem to be fulfilled, and we must then suppose that eddies are produced in the blood which will cause sound-vibrations; in others, again, the sound may be due to vibration of the edges of the valve.

Murmurs differ from one another—(1) in time; (2) in their relation to the orifices of the heart; (3) in the character of the sound.

(1) Murmurs which are heard with the first sound, or between the first and the second sounds, occur during the contraction of the ventricles, and are called *systolic;* those which are heard with the second sound, or between it and the succeeding first sound, occur during the dilatation of the ventricle, and are called *diastolic.* Amongst these last, one can distinguish some which begin at the very commencement of diastole and end before the next first sound; others, which commence a little later, but still end before the first sound; and others, again, which begin after the second sound, and run up to, and finish, in the next first sound. Bristowe called these respectively *early*, *mid*, and *late* diastolic. The

last is more commonly known as *presystolic*. In determining the rhythm of a particular murmur, its position should be noted in reference to the beat of the heart, or to the beat of the carotid artery by the side of the thyroid cartilage. Either of these represents the systole of the ventricle with sufficient accuracy, but the radial pulse is a little later.

(2) It is sufficient to say here that murmurs of systolic and diastolic time may be heard, with varying frequency, in connection with all four orifices of the heart. The subject will be more fully discussed in the chapter on "Diseases of the Valves."

(3) The quality of the sound is most often blowing; it is sometimes rushing, sawing, or rasping. Sometimes murmurs have a distinctly *musical* quality. Half-detached fragments of valve playing in the blood-current, perforations in valves, and loose chordæ tendineæ sometimes cause such murmurs.

THE ARTERIAL PULSE.

By the term *pulse* is meant the movements of alternating expansion and contraction which are felt in any artery of the body accessible to the finger. These serve as an important means of ascertaining the action of the heart and the condition of the circulation. The artery which is mostly employed for this purpose is the radial artery at the wrist; but a pulse can also be felt with no difficulty in the ulnar artery at the wrist, in the brachial in the arm, in the carotid by the side of the thyroid cartilage, in the facial artery as it turns round the lower jaw, in the temporal artery above the ear, in the femoral artery below Poupart's ligament, in the posterior tibial behind the inner malleolus, and in the dorsalis pedis at the base of the metatarsal bones.

It should be remembered that the radial artery does not always lie in its normal situation, but sometimes turns over the radius to the back of the wrist, one or two inches above the joint; and this may happen on one or both sides. A minute communicating branch may sometimes be felt in its place; but, in any case, the absence of a pulse of the proper size may be very misleading unless the occasional abnormality be borne in mind. More rarely, the radial is abnormally small, and the comes nervi mediani compensates for this by its unusual size.

In examining the pulse, one has to observe its *rate*, and its *volume*, and other characters about each individual beat, especially its *hardness*.

The Pulse-rate.—Normally the heart beats about seventy-two times in the minute, with variations in health between fifty and eighty.

Excessive slowness of the pulse occurs in some cardiac lesions, especially aortic stenosis and fatty heart; in conditions of collapse, in meningitis and cerebral tumours, and in jaundice. Occasionally the pulse appears to be slow because some of the heart's beats are not powerful enough to drive a wave so far as the wrist. These feeble beats generally alternate with stronger beats; so that while the heart contracts, say eighty times in the minute, the pulse is only forty. An intermediate condition is where there are indeed as many pulse-beats as cardiac contractions, but every alternate pulse-beat is so feeble as to be only just perceptible (*pulsus alternans*).

Increased frequency of the pulse is much more common in disease than great slowness. Nearly every disturbance of health tends to quicken the pulse; it is the constant accompaniment of acute and subacute inflammations, of fever in all forms, of states of exhaustion, and of most forms of cardiac disease.

Irregularity, or Arrhythmia.—The intervals between successive beats of the pulse should be of uniform length; when this is not the case the pulse is irregular. The irregularity may be of such a kind that no order can be observed at all; long, short, and shorter intervals follow each other indiscriminately. This occurs commonly in mitral disease, in dilatation of the ventricle, and, in a less degree, in some cerebral lesions—*e.g.*, meningitis.

Another form of irregular pulse is the *intermittent* pulse. Here several successive beats come quite regularly, and then the heart seems to stop, and an interval occurs which is about equal to a whole revolution of the heart. The heart is said to *intermit*. The pulse again beats regularly for a time, and another intermission occurs. The intermissions are frequent, viz., every four or five beats; or more rare, viz., every twenty or thirty; but the number of beats between the intermissions is not itself absolutely uniform. An intermittent pulse is sometimes the result of cardiac disease, sometimes a purely functional or temporary disorder.

Another form of irregularity is known by the names *pulsus bigeminus*, *twin-pulse*, *coupled-beats*, and *allorrhythmia*. In this the beats occur in successive pairs, with a longer interval between any two pairs than between the beats of each pair. This occurs in some cases of mitral disease, and is occasionally induced by the use of digitalis. It may be quite temporary, or, at any rate, it may rapidly change into a perfectly regular beat, and again recur in the course of a few hours. A triple beat (*pulsus trigeminus*) is sometimes observed under similar circumstances.

Pulsus paradoxus is of rare occurrence. It consists in the diminution or complete disappearance of the pulse during inspiration, and is explained by inspiration bringing into action some cause of pressure upon the aorta, such as bands of adhesion.

The Volume of the Pulse.—This is determined partly by the actual size of the artery, partly by the quantity of blood sent into the artery at each beat of the heart. If much blood is sent in, the pulse is full or large; if but little blood, the pulse is small.

Hardness of the Pulse: Arterial Tension.—If the finger be placed upon the radial artery, and pressed upon it with increasing force, the flow of blood is at length stopped; and this arrest of the blood-flow can be more easily effected in some pulses than in others. Those in which slight pressure is sufficient are called *soft* or *compressible* pulses; those in which much pressure is required are called *hard* or *incompressible* pulses. If when the pulse has been stopped by compression the finger be slowly lifted, the blood will be felt to pass under the finger with much greater force in the case of the hard than in the case of the soft pulse; and the estimation of the hardness of the pulse is generally much helped in this way. A hard pulse is not necessarily very small or very large. It feels like a cord, but it is important to distinguish from a purely hard pulse that kind of rigidity which is due to atheromatous and calcareous changes in the wall of the artery. The wall then feels rough and irregular, and the vessel has often a tortuous course. The two conditions, rigidity of arterial wall and hardness of pulse, may co-exist. A pulse is hard in proportion to (1) the quantity of blood thrown into the arterial system, (2) the difficulty of egress through the arterioles, capillaries, and veins (often spoken of as capillary resistance), and (3) the degree of contraction of the arterial coats upon their contents. It is soft under the opposite conditions. Thus, hardness is favoured by a powerfully acting heart, a normal amount of blood, contraction of the peripheral arterioles—as, for instance, by cold, which stimulates the muscular coat of the arteries (vasomotor stimulation). Softness of pulse is favoured by a feeble heart, by valvular imperfections interfering with the supply of the blood to the arterial system, by a free flow through the capillary area, and by dilatation of the arteries and arterioles as a result of vasomotor paralysis. The hard pulse is indicative of *high arterial tension;* the soft pulse of *low arterial tension.* The former is often accompanied by an accentuated aortic second sound, heard at the inner end of the second right intercostal space, and sometimes by a reduplication of the first sound, heard over the septum ventriculorum (p. 493).

Dicrotism.—Especially in febrile conditions, but also in the soft varieties of healthy pulses, one can feel a doubling of each pulse-beat, which is known as dicrotism. As a rule, the main beat is followed by a much slighter elevation of the artery-wall. In most pulses there is no difficulty in distinguishing this from a true pulse-beat, because it takes place in the course of the elevation due to the ventricular contraction, and auscultation proves that there is no ventricular systole corresponding to it. It is due to a wave reflected from the closed aortic valves and the aortic walls, and transmitted peripherally to the wrist. It is favoured by the conditions producing softness of pulse, and is most marked in highly febrile stages; it is diminished by conditions leading to hard pulse, such as Bright's disease, and by aortic regurgitation, in which case the reflection of a wave takes place imperfectly.

The Sphygmograph.—The accurate estimation of the pulse is much aided by the sphygmograph, which, at the same time, furnishes a permanent record for future comparison.

The needle of the sphygmograph makes a tracing on blackened paper, and each beat of the heart is shown by the needle rising above the base line, and again falling. The frequency or regularity of the pulse can thus be seen at a glance; but the special features of each beat are of more importance. In the tracing of each beat of the arterial pulse there is an upstroke, which is uninterrupted and almost vertical; and a downstroke, which is oblique, and interrupted by one or two elevations with intervening depressions.

The upstroke represents the contraction of the ventricle, driving blood into the aorta and thereby causing a wave which is rapidly transmitted to the peripheral arteries. The apex of this upstroke has been known as the *percussion-wave*. Its height is proportionate to the force of the ventricular contraction, and the quickness or suddenness of the contraction is indicated by the vertical course of the stroke. The height is also greater when the arterial wall is yielding, less when it is tense and resistant. Compare Figs. 20 A and 21 A, B with Figs. 20 B and 22 A, B, C.*

Of the elevations in the course of the downstroke, the most constant is the *dicrotic wave* (Fig. 20 A, *c*, Fig. 21 A, *c*, Fig. 22, A, *c*). This is the same as can be felt with the finger in pulses that are called "dicrotic"; it is shown by the sphygmograph to be present in the majority of pulses, even when not perceptible to the finger. As already stated, it is due to a reflected wave from the closed aortic valves and from the walls of the aorta. It is immediately preceded by a depression, the *aortic notch*, the depth of which is proportionate to the size of the dicrotic wave. In Figs. 20 and 22 they are both slight, in Fig. 21 they are both well marked. When the aortic notch reaches the base line (Fig. 21 A) the pulse is called *fully dicrotic;* it sometimes falls below the base line, and is then called *hyperdicrotic* (Fig. 21 B). In this case the percussion wave of the next beat appears to come before the dicrotic wave has completely passed. Dicrotism is best marked in soft pulses, with yielding and elastic walls; it is a common result of vasomotor paralysis as seen in highly febrile conditions (Fig. 21), and can be at once produced by the administration of amyl nitrite. The aortic notch corresponds to the termination of the systole of the ventricle —that is, it marks the closure of the aortic valves.

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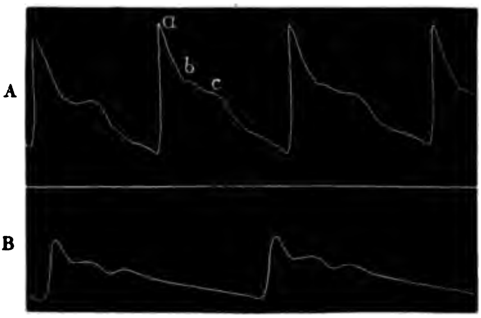
* The tracings were taken with a Marey's sphygmograph; a long and quick upstroke is curved backward, because the needle is at the end of a long lever, which works on a fulcrum, with an axis transverse to the line of movement of the paper.

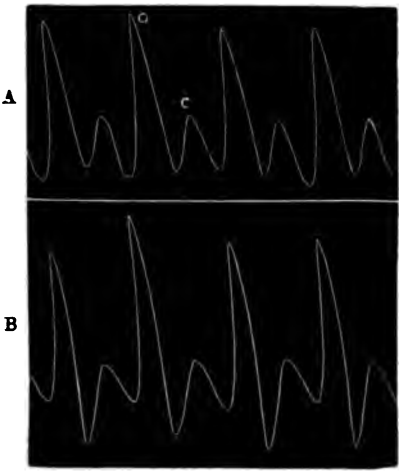
Between the percussion wave and the dicrotic wave—that is preceding the aortic notch, and therefore corresponding to the period of systole of the ventricle—there is often a wave which has been attributed to the outward flow of the current of blood following the percussion wave. It is called the *tidal* or *predicrotic wave* (Fig. 20 A, *b*, Fig. 22, A, *b*, B, C). It is best seen in hard

FIG. 20.



A. Normal Soft Pulse. Pressure Two Ounces.
B. Hard Pulse in Gout.

FIG. 21.



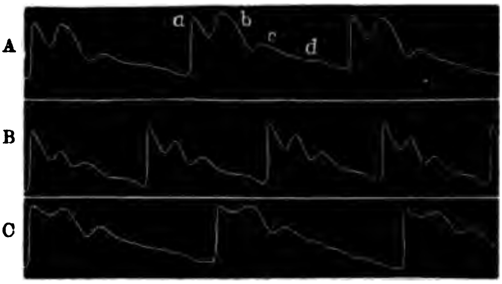
A. Dicrotic Pulse in Pyrexia. Temp. 102·2°.
B. Hyperdicrotic Pulse in Pyrexia (Enteric Fever). Temp. 103°.

pulses (Fig. 22)—that is, in conditions of high arterial tension, when it may be supposed that the undulations of the blood would be unusually well transmitted. On the other hand, in very soft pulses, the tidal wave is lost in the percussion wave (Fig. 21, A, B). A pulse, in which the tidal wave rises higher than the percussion wave, has been called *anacrotic*, because the percussion wave forms an elevation in the ascending limb between the base and the highest point.

One or two slight undulations are occasionally seen after the dicrotic wave (Fig. 22 A, *d*). They occur in tracings of pulses of high tension only.

Landois describes only two primary waves in a sphygmographic tracing—namely (1) that which we have called the percussion wave, and (2) the dicrotic wave. He calls the tidal wave and the undulations which follow the dicrotic wave, ***elastic elevations***, and regards them as being secondary to the percussion and dicrotic waves respectively, in consequence of the high tension in the vessels.

FIG. 22.



A. Acute Bright's Disease. Pressure, Four Ounces.
B. Acute Bright's Disease; Five Weeks' Duration. Pressure, Seven Ounces.
C. Chronic Bright's Disease. Pressure, Six Ounces.

Whatever be the explanation, it remains the fact that conditions of high arterial tension are indicated by a high tidal wave, and by moderate dicrotism; whereas in low tension the dicrotism is considerable, and the tidal wave is small or absent.

It has been already said that the pulse of high tension is hard, and resists compression by the finger. The amount of this resistance can be accurately estimated by a properly constructed sphygmograph, in which a graduated pressure can be applied to the spring, which lies upon the artery. It will then be ascertained that some very hard pulses require a pressure of twelve or fourteen ounces to stop the flow of blood in them; while in others the current can be arrested by a pressure of five or six. Similarly, in the former kind of pulse a pressure of five, six, or seven ounces is necessary in order to get a correct tracing, whereas in soft pulses a pressure of one, two, or three ounces is sufficient (compare Fig. 22 with Fig. 20 A).

Certain valvular diseases and some alterations in the walls of the vessels impart special features to the pulse, which will be dealt with in future chapters. The pulses which result from mitral disease, from aortic regurgitation, from arterial obstruction, from aneurysm, and from atheroma, are the most characteristic (Figs. 27, 28, and 29).

When the heart's action is very feeble, the base line of the tracing may be undulating instead of straight. This shows the incapacity of the ventricle to overcome the influence of the respiratory movements upon the circulation, and is called the *respiratory wave.*

THE VENOUS PULSE.

Some pulsation in the large veins of the body appears to be a normal phenomenon; and an undulating or distinctly pulsatile movement can be seen in the jugular veins, especially the *external*, of many persons with quite healthy circulatory organs. The expansion of the vein is then synchronous with the dilatation of the ventricle (cardiac-diastolic), and its collapse with the contraction of the ventricle (cardiac-systolic).

Another kind of pulsation, in which this relation is reversed, takes place in tricuspid regurgitation; the expansion of the vein-wall is synchronous with the contraction of the right ventricle, which forces a wave of blood into it through the leaking tricuspid valve; the contraction of the vein results when the ventricle relaxes. This cardiac-systole pulsation or expansion is seen mostly in the *internal jugular* vein, and the patient so affected shows an extensive undulating movement of elevation and subsidence at the side of the neck between the ears and the clavicle, and posterior to the site of the carotid artery. The external jugular may pulsate at the same time. A tracing from the jugular vein shows an elevation in the ascending limb (*anacrotic*), and an unbroken line of descent.

In tricuspid regurgitation the effect of the right ventricular contraction may be transmitted also to the hepatic veins in the liver, so as to cause *hepatic venous pulse*, or *pulsating liver*. The organ is commonly much enlarged, and can be felt throbbing over its whole surface; and the pulsation is sometimes even conveyed behind into the right loin under the last rib, so that the organ can be felt to expand between the hands placed in front and behind.

Pulsation is sometimes seen in the peripheral veins, especially those of the backs of the hands and feet, which is due to the transmission of the arterial wave through the capillaries to the veins.

It results from great relaxation of the vascular walls, with a powerful or excited action of the heart; thus it may occur in febrile conditions, in the heat of summer, or after full meals.

DISEASES OF THE HEART.

ENDOCARDITIS.

Endocarditis, or inflammation of the lining membrane of the heart, shows itself in two forms—*simple* and *malignant*—but they are connected by intermediate links.

SIMPLE ENDOCARDITIS.

This is commonly described as acute or chronic; but the term chronic endocarditis is often applied to the permanent deformities and alterations in the valves, which result from acute endocarditis, as well as to a separate inflammatory process of slow development.

Ætiology.—Endocarditis is, in the great majority of cases, caused by rheumatic fever; it also occurs during the progress of chorea, and in scarlet fever, diphtheria, typhoid, and some other infectious diseases. Bright's disease, syphilis, and other chronic dyscrasiæ, are said occasionally to cause it. It may be brought on by local injuries, such as the rupture of a sigmoid valve, or of the chordæ tendineæ, and the unnatural friction of one part of the heart with another; and the passage of currents of blood through abnormal apertures may cause the local inflammation of the endocardium.

It is always in limited patches, and never affects the whole interior of the heart. In rheumatism and other general diseases it affects the valves first or alone.

The relation of endocarditis to the two sides of the heart is of very great importance. Endocarditis occurring during fœtal life, and the malignant form to be presently described, may attack the pulmonary or tricuspid valves. But in other cases, simple acute endocarditis is almost invariably on the *left* side. Hence what follows in this chapter chiefly concerns the aortic and mitral valves.

Anatomical Changes.—The earliest change is a very slight swelling of the subendocardial tissue along the margin of the valve which is opposed to its fellow, and touches it on closure of the orifice. This swelling results from œdema and infiltration with leucocytes, and is commonly seen in the form of a number of bead-like elevations, described usually as *vegetations*. These may entirely subside, but if they persist they become covered with deposits of fibrin, which intimately unites itself with the deeper tissue containing the leucocytes. By the continued addition of deposits of fibrin, very large vegetations may be formed which project into the valvular orifice, and from which particles, loosened by softening, may be detached by the force of the current of blood. Such a detached particle is carried along with the blood-current to distant vessels of gradually diminishing size, and ultimately meeting with one small enough to resist its further progress, becomes impacted therein. This process is known as *embolism*: it is much more frequent in malignant endocarditis. At different stages short of this, subsidence of the inflammation may occur; and though in the earliest period complete resolution probably may take place, there is more often some organisation of the leucocytal infiltration, and fibrous tissue forms, by the gradual and irregular contraction of which—a process similar to that which occurs after inflammation in other parts—the valves become shortened, deformed, and incapable of completely covering the orifice they are intended to close. In some cases the fibrous tissue acquires an almost cartilaginous hardness, or calcareous particles are deposited, and the valve segments not only fail to close the valve aperture, but, by their constant projection into the orifice, offer a definite obstruction to the passage of the blood through it.

In acute stages, streptococci, staphylococci, and pneumococci may be found; but they are absent from the chronic lesions.

Symptoms.—Endocarditis, as it occurs in acute rheumatism, has but few symptoms. Indeed, it mostly proceeds without any appreciable alteration of those which are due to the rheumatism, and is detected only by the stethoscope. It happens in somewhat less than half of all cases of rheumatic fever, and mostly within seven days of the beginning of the attack. If the heart be carefully auscultated, the first indication will be a slight prolongation, or roughness, or some want of clearness of the first sound at the aortic or mitral area, according as the one or the other valve is the seat of inflammation. Within twenty-four hours it may lengthen into a distinct murmur, or soft blowing sound, which accompanies, and does not abolish, the first sound. If the aortic valve is affected, the second sound may become imperfect, and a diastolic murmur may become developed, but this is much less frequent, while the systolic murmur in the mitral area is the most common of all. As above stated, this physical sign may be the only indication of endocarditis, but increased force and frequency of the heart's action, palpitation, and some præcordial pain and distress are occasionally observed. The evidences of endocarditis persist for a variable time; the murmur may disappear entirely in the course of the rheumatic attack; or it may become louder and harsher, more widely diffused, or definitely follow the course of the blood-current, showing the existence of valvular obstruction or incompetence.

Diagnosis.—This requires some care, as the murmurs of recent acute endocarditis may be confounded with *functional* murmurs, with the murmurs of old *valvular disease*, and with *pericardial* friction sounds. The chief point to note is that the murmur of acute endocarditis is generally soft in quality, systolic in time, and strictly limited to the area of the valve affected—that is, either the aortic or the mitral area. Acute simple endocarditis of the pulmonary or of the tricuspid valve is practically out of the question. A functional or hæmic murmur is generally loudest over the pulmonary artery, and often harsh in quality. The murmur of chronic valvular disease is often loud or harsh, heard over a large area, and accompanied by some alteration in the size or shape of the heart.

Prognosis.—In the course of an attack of rheumatic fever, or of any other illness causing acute simple endocarditis, there is nothing to guide one as to the outcome of the disease. In a large number of cases the murmur disappears, and the patient apparently recovers completely; in a certain proportion of these, nevertheless, valvular disease supervenes several years afterwards. In a few cases the affected valve soon becomes incompetent, and the patient suffers henceforth from "heart disease."

Treatment.—The influence of treatment upon acute simple endocarditis is not very apparent, and many are content to leave it alone. Sometimes a small blister, about three inches by two inches, is placed on the chest over the affected valve. The treatment proper to acute rheumatism should be continued, and this will, of course, necessitate rest in the recumbent position and light diet, which are also desirable for endocarditis.

MALIGNANT ENDOCARDITIS.

(*Septic, Infective, or Ulcerative Endocarditis.*)

Ætiology.—The connection of this form of endocarditis with rheumatism is by no means so intimate as that of simple endocarditis, although in a certain proportion of cases (53 out of 160—Osler) a rheumatic history was obtained: in some of these the symptoms have developed in the course of the rheumatic fever, and in others they have arisen in the stage of chronic valvular disease, which has an important influence in the occurrence of infection. Malignant endocarditis may occur quite spontaneously—at any rate, without any previous history to explain it in the present state of our knowledge. On the other hand, besides rheumatism, its predisposing cause may be found in acute pneumonia, in the eruptive fevers such as scarlatina, in puerperal processes, in the existence of open wounds on the surface of the body, in purulent discharges from the mucous membranes (urethritis, vaginitis), in septicæmia and pyæmia, in ague, and in some other conditions. Various micro-organisms are found in infective endocarditis. They are the pyogenetic microbes, streptococcus pyogenes, staphylococcus pyogenes, and pneumococcus, which are most common; the bacillus pneumoniæ of Friedländer, and the bacilli of tubercle, diphtheria, and typhoid. In addition there are found organisms which are not present in other diseases.

From some centre of infection, the organisms find an entrance into the blood and are thence deposited on the valves.

Anatomical Changes.—In this form of endocarditis the tissue of the inflamed valve is softened, and breaks down, so that erosions or ulcerations take place, and, as a result of this, fibrin is deposited upon the roughened surface, and accumulates into irregular masses of vegetations, which may reach the size of a hazel-nut. By the use of suitable colouring agents the micro-organisms can be demonstrated on the surface, and more or less deeply in the substance of the vegetations and fibrinous deposits, forming considerable masses, or colonies. Several important changes result from these processes in the valve. The valve itself may be perforated, or strips of tissue may be partly separated and hang loosely in the blood-current, or portions may be completely detached. Sometimes a part of the valve is so weakened by the destructive process that it yields before the pressure of the blood, and a saccular dilatation, or *aneurysm*, of the valve is formed, projecting on the opposite side. Another result is the occurrence of endocarditis, or endarteritis, in adjacent parts from a strip of the valve playing backwards and forwards in the blood-currents with the systole and diastole of the ventricle, and striking alternately the walls of the cavities in front and behind. In the case of mitral endocarditis, these are the left ventricle and the left auricle; in the case of aortic endocarditis, they are the aorta and the left ventricle. At the spot struck infection takes place, and causes a fresh patch of inflammation of the lining membrane.

But the most important effect of malignant endocarditis is the infection of the whole arterial system by particles detached from the valves being carried to remote parts; and it is to this process, combined with the presence of organisms in the detached fragments, that the special features of this disease are due. Embolism takes place in the most various parts of the body. It is especially common in the vessels of the spleen and kidneys, but it happens also in the vessels of the brain, alimentary canal, skin, retina, and lungs, and the larger vessels supplying the limbs, such as the radial, ulnar, tibial, brachial, and others. The local results of these impactions are:—(1) Obstruction of the circulation; (2) hæmorrhage within the area of distribution of the obstructed vessel, as seen in the infarcts of the spleen and kidneys; and (3) suppuration in the same area from the septic influence of the micro-organisms.

The effects upon the various organs, as they may be seen in different cases of malignant endocarditis, are—softening and abscess of the brain, and meningitis; retinal hæmorrhages and optic neuritis; diffused swelling, hæmorrhagic infarctions, and abscess of the spleen; hæmorrhagic infarctions, or general diffused inflammation of the kidneys, which are often large, and finely mottled or speckled with hæmorrhagic points upon a white ground; hæmorrhages under the skin; hæmorrhagic infarctions and abscesses of the lungs; pleurisy and empyema (*see also* Embolism).

Malignant endocarditis, like simple endocarditis, affects chiefly the left side of the heart; but the proportion of cases in which the right side is involved is much larger than in the simple form.

Symptoms.—Two varieties of the disease are commonly recognised, though some cases combine the characters of each, and others do not strictly conform to either. These forms are the *typhoid* and the *pyæmic.* In the *typhoid* form, the patient may have been perfectly well until he complains of some such symptoms as usher in other severe febrile diseases, pain in the head, or in the back and limbs, or a definite rigor or rigors. Then follows severe pyrexia, with its usual conditions—high temperature, quick pulse and respiration, dry tongue, loss of appetite, thirst, and malaise. Frequently, within a few days, the patient is prostrate, apathetic, drowsy, and at night delirious; but the time of appearance of this symptom is determined, as in typhoid fever, by the severity of the disease. The temperature is high, reaching 103°, 104°, or 105°, but is generally remittent or intermittent, and sometimes with remarkable regularity for long periods. There is often free perspiration. The pulse is rapid, ranging from 100 to 140, or even higher. If the heart be auscultated, a murmur will generally be heard at one or other orifice, mostly, however, on the left side. Still, it must not be forgotten that in these cases murmurs may be entirely absent. In cases with a previous history of rheumatism, there may be more complete evidence of cardiac disease in enlarged præcordial area, displacement of the impulse, or irregular action. The respirations are rapid, sometimes without definite lesions of the lungs, at others with signs of bronchitis, œdema, or congestion. The condition of the bowels varies, but there are often loose, yellow motions, with much resemblance to those of typhoid fever; and the abdomen may be distended.

The embolism of large or small arteries in different parts of the body leads to important symptoms. Sometimes embolism of a large vessel in the brain may lead to hemiplegia, or if in a limb it may cause loss of pulse at the wrist or in the leg, and be followed by gangrene; but embolisms of these larger arteries are not so frequent as those of the small vessels in the viscera. There is thus frequently enlargement and tenderness of the spleen due in part to infarcts; and infarcts occur in the kidney accompanied, it may be, with pain, and the appearance of albumen or blood in the urine. In some cases petechial hæmorrhages appear under the skin, the petechiæ being generally small, and situate on the trunk, about the groins and axillæ. Hæmorrhages are often seen in the retina, and there may be also hæmoptysis or epistaxis. Some inflammatory conditions are probably also referable to embolic processes, such as the form of nephritis already mentioned (*see* p. 505), though whether as the result of vascular obstruction or of the introduction of micro-organisms may be doubtful. Albuminuria from nephritis or infarct is frequent; and another important sign is a pronounced anæmia, which occurs even when there has been no hæmorrhage. Optic neuritis is sometimes present. The tongue shows the usual changes of febrile diseases, being at first moist, with white fur, subsequently dry, glazed, or brown and cracked. In the more advanced stages, low delirium is mostly present, at first by night only, later on continuously, and this may lapse into complete coma before death.

In the *pyæmic* form rigors are a prominent symptom, occurring once, twice, or more times in the day. Rheumatism and actual cardiac disease are less often present as antecedents, and the endocarditis more often affects the right side of the heart than in the typhoid form. It may begin, like the last, with vague or more decided pains in the limbs or back, until the first rigor occurs; and intense anæmia is often present. The heart may present but little or no evidence of enlargement, and the murmur may be limited to the area of the pulmonary artery, in which case doubt will arise as to whether the murmur is simply hæmic, as a consequence of the pronounced anæmia; on the other hand, in many cases the same conditions of the cardiac apparatus may exist as in the typhoid form—namely, the murmurs of mitral or of aortic disease. For the rest, the local conditions are not very different from those of the typhoid form. The spleen may be enlarged, diarrhœa with loose yellow motions often occurs, and albuminuria may be present. Sometimes the joints inflame or suppurate. The temperature rises to a great height, 105° or 106°, in the rigors, and may fall to the normal or subnormal in the intervals. Sweating occurs, and is often profuse. As the case continues, emaciation becomes more marked, and with increasing delirium and apathy or coma death results.

A special character is given to some cases by the complication of pneumonia, pleurisy, or meningitis; and it is believed that the particular valves involved and the nature of the complication may depend upon the variety of bacillus causing the disorder: that is, upon the antecedent disease, when there has been one. Thus, it has been noted that suppurative meningitis occurs in cases preceded by pneumonia.

The duration of malignant endocarditis is very variable. It may be fatal in two or three weeks, or may last six or seven months, with pyrexia throughout. In cases of old heart disease, the supervention of this form of endocarditis is indicated by remittent pyrexia, and by the occurrence of embolism in different parts of the body, but the cardiac symptoms may continue predominant, and thus distinguish this group of cases from those first described.

Diagnosis.—This rests upon the pyrexia of remittent or septic type, the existence of valvular disease, and the evidences of embolism above enumerated. Marked anæmia and optic neuritis, if present, are also valuable signs. Streptococci have been discovered in the blood during life. *Typhoid fever* is the disease most likely to be confounded with it—that is, malignant endocarditis is frequently mistaken for typhoid fever. Both are acute severe febrile diseases, and in both there may be a marked absence of localising symptoms; on the other hand, there is often in malignant endocarditis swelling of the abdomen, with frequent loose yellow motions, and enlargement of the spleen may further increase the resemblance. The following are points of difference:— In malignant endocarditis there is a more uniformly remittent or intermittent course of temperature, which may last several weeks; rigors, petechiæ under the skin, much pallor of the face, optic neuritis, or retinal hæmorrhages, if present, are more in favour of endocarditis; and a cardiac murmur, especially if it is observed to change its characters from time to time, is strong presumptive evidence.

The diagnosis from *pyæmia* depends largely on the presence of the cardiac murmur. Generally, the pyæmic patient has more frequent rigors, an earthy colour of the face, and the evidences of inflammation at the base of the lung. The presence of a wound is not distinctive, as it may be a cause of septic endocarditis; and the diagnosis is further complicated by the fact that the endocarditis may itself be only a part of pyæmia. *Ague* may be closely simulated by endocarditis, with even quite regular attacks of pyrexia; the absence of malaria as a cause, and the inefficacy of quinine in the treatment may help to a diagnosis. A continued pyrexia without obvious signs may be due to commencing *miliary* *tuberculosis* as well as malignant endocarditis; but after a time local signs peculiar to one or the other ought to be observed.

The development of malignant endocarditis in rheumatism, pneumonia, or old valvular disease must be recognised by the typhoid or septicæmic type of the symptoms, and by changes in the cardiac sounds indicating recent lesions.

Prognosis.—This is exceedingly bad; and the recovery of a well-marked case of either typhoid or pyæmic form is very rare. On the other hand, attacks of pyrexia in old valvular disease, with or without evidence of embolic processes, have subsided sometimes, to occur again after an interval of weeks or months.

Treatment can obviously be little more than palliative in the majority of cases. As in pyæmia, if there is any wound or sore, it should be rendered aseptic, and an attempt may be made to influence the disease by frequent doses of quinine (5 gr.), sodium sulphocarbolate (10 to 20 gr.), or sodium benzoate (20 gr.). In a few cases good results have followed the subcutaneous injection of an antistreptococcus serum. The general rules for nursing and dieting in typhoid fever are applicable here: milk, beef-tea, and other light nutriment being given frequently in small quantities. Profuse diarrhœa may be checked, if required, by astringents. The delirium is rarely so violent as to require any special treatment. Stimulants are naturally given, as the heart's action early tends to be seriously affected.

CHRONIC ENDOCARDITIS AND DISEASES OF THE VALVES.

As a result of the several changes in the structure and shape of the valves produced by endocarditis, their efficiency is seriously impaired, and the circulation of the blood through the heart is to a greater or less extent affected. This takes place in two ways: first, the thickening of the valve, or vegetations or fibrinous masses upon it, or the union of two or more segments of a valve together, may materially narrow or constrict the orifice through which the blood passes from auricle to ventricle, or from ventricle to aorta. Secondly, the contractions or deformities of the valves may so shorten them, or diminish their area, as to render them incapable of closing the orifice, and preventing reflux of blood from ventricle to auricle, or from aorta to ventricle. The one case is called *obstruction* or *stenosis*, the other *regurgitation* or *incompetence*. These two conditions may occur singly or combined at any one of the four orifices of the heart; but they are much more frequent on the left side of the heart than the right, as endocarditis, which is the chief cause of the valvular deformities, rarely attacks the right heart. It must, however, be remembered that regurgitation may arise from a simple dilatation of the orifice, which the valve is unable to cover, although itself healthy. This takes place frequently in the case of the tricuspid and mitral orifices, and accounts for many instances of the tricuspid regurgitation which follows upon disease of the left side of the heart. At the aortic orifice ruptures of the segments of the valves from strain and injury will lead forthwith to regurgitation, while secondary endocarditis and deposits of fibrin upon the injured valve will lead to obstruction in addition. At the pulmonary orifice, organic obstruction and regurgitation are mostly the result of inflammatory changes during fœtal life, which hinder the development of the heart, and produce congenital malformations, with a special train of symptoms (*see* Congenital Malformations); but they are sometimes due to malignant endocarditis of the pulmonary valves (*see* p. 506).

Relative Frequency of Valvular Lesions.—Mitral valve disease is more common than aortic valve disease. At the mitral orifice, regurgitation alone is the most frequent, a combination of obstruction and regurgitation next in frequency, and pure obstruction least frequent. At the aortic orifice, double disease (obstruction and regurgitation) is most common, simple regurgitation comes next, and pure obstruction is comparatively rare. Mitral regurgitation is often the result of, and then accompanies, aortic disease. On the right side of the heart, tricuspid regurgitation is the only form that is at all frequent, and it is mostly secondary to mitral disease, or to chronic lung disease, such as emphysema or bronchiectasis. It may follow mitral disease, when this is the result of aortic disease.

Effects upon the Heart.—The ordinary phenomena and symptoms of valvular diseases of the heart are the effects, direct and remote, of the obstruction and regurgitation which accompany them. Valvular disease may occasionally exist for even long periods without producing any apparent result, except the physical sign of cardiac murmur, by which its presence is detected. The reserve power in the heart is sufficient to overcome the slight obstruction which the valve disease has created. But sooner or later the obstruction at the orifice, or the imperfect action of the valve leads to a structural change in the walls of the heart. In accordance with what takes place in other parts of the body, the increased work thrown upon the walls of the left ventricle, in order to overcome an obstruction existing at the orifice of the aorta, leads to its *hypertrophy*, so long as it is adequately nourished, and up to a certain limit, which, no doubt, varies under different circumstances. If the resistance in front be carried beyond this limit, or if the muscle of the heart be insufficiently nourished, then the walls of the cavity will yield before the increased pressure they are subject to, and *dilatation* will take place. Hence, according to circumstances, valvular lesions may result in hypertrophy alone, or dilatation alone, or both together; and the last condition is the most common.

When the valvular lesion is so slight as to be adequately met by the reserve power of the heart, or where hypertrophy has developed sufficiently to overcome the difficulty, the lesion is said to be *compensated:* in the converse condition, when the heart is giving way before the lesion and dilatation is in excess, the disease is said to be *uncompensated.*

Hypertrophy and dilatation affect primarily the walls of the cavity immediately behind (in the course of the circulation) the valve which is diseased—that is, the left ventricle is first affected in disease of the aortic valves, and the left auricle in obstruction of the mitral orifice; but the changes are not limited to these cavities respectively. For, first, by continued dilatation of the left ventricle the mitral orifice will become enlarged, and regurgitation will take place in the way above mentioned, and then the left auricle will be placed in the same position of overstrain with regard to the mitral orifice, as was the left ventricle with regard to the aortic orifice. And, secondly, without actual dilatation, the pressure in the circulation behind the first affected cavity will be so increased as to involve successively not only the cavities of the heart, but also other structures through which the current of the blood passes. In this way, disease of the aortic valves may be followed successively by dilatation and hypertrophy of the left ventricle, the left auricle, the right ventricle, and the right auricle; and constriction of the mitral orifice successively by similar changes in the left auricle, the right ventricle, and the right auricle.

Congestion of the lungs, which lie, as it were, between the left auricle and the right ventricle, contributes to the effect upon the right side of the heart. It increases the pressure in the pulmonary artery, which the right ventricle may for a time overcome, but ultimately the wall of this cavity yields, and the tricuspid valve becomes inefficient. This may happen either from dilatation of the orifice itself, or from the distension of the ventricular walls drawing down, away from the plane of closure, the segments of the tricuspid valve which are attached to them by the chordæ tendineæ and the papillary muscles. The delay is then felt in the right auricle, and since this receives the blood of the superior and inferior venæ cavæ, the whole of the systemic venous circulation is thereby affected.

The size and shape of the heart are much altered by these changes, and differently according to the cavity or cavities concerned. When the *right ventricle* is much dilated, the triangular shape of the heart is lost, it becomes more globular, and the apex of the heart is formed partly by the right ventricle instead of being formed entirely by the left. With hypertrophy of the *left ventricle*, the enlargement tends to be in a vertical direction, so as to displace the apex of the heart downwards; but when dilatation is in excess of hypertrophy, there is also great enlargement laterally, and the impulse may be felt, during life, four or five inches to the left of its normal position. The walls of the left and right ventricles may reach double their normal thickness; but the walls of the auricles, though dilating considerably, do not become thick in proportion. As a result of these changes the weight of the heart may increase from the nine, ten, or eleven ounces which are normal, to sixteen, eighteen, or twenty, according to the number of the cavities involved and the extent of hypertrophy. The greatest enlargement occurs in aortic disease, in which the heart has been known to attain the weight of forty-eight ounces. Such examples are known as *bovine* hearts.

Fibroid and fatty degeneration of the myocardium are sometimes the results of chronic valvular disease.

Effects upon other Organs.—The secondary remote effects of valvular disease on other organs, produced as they are by defects in the circulation of the blood which pervades all parts of the body, are numerous and widespread. They are most manifest in the subcutaneous tissues, which become œdematous, and in the lungs, liver, kidneys, spleen, and gastro-intestinal mucous membranes. The most frequent of these are *venous congestion* and *œdema*.

Anasarca.—A general œdema of the subcutaneous tissues is called anasarca. In heart disease it begins in the feet and ankles, and gradually extends up the legs. But even when it is very extensive it is generally confined to the lower extremities and lower half of the trunk, leaving the face, chest, arms, and hands of their normal size.

Lungs.—The delay in the circulation which results from impaired action of the valves on the left side of the heart first affects the lungs; the blood in the pulmonary veins flows with difficulty into the left auricle, and more or less stagnation of the blood in the capillaries takes place. In early stages there is simply undue fulness of the venous radicles in the lung; there is often, in addition, a transudation of serum into the air-vesicles and minute bronchial tubes, so that on section of the lung a quantity of yellowish or almost colourless frothy liquid flows from it; and in advanced cases the most affected parts of the lung become solid, tough, airless, dull red in colour, and uniformly smooth, resembling somewhat the cut surface of the spleen. This condition has been called *splenisation*, *heart-lung*, or *red induration*, and appears to be a mixed condition of œdema and congestion with minute hæmorrhages. In a later stage, the colour is browner from the presence of pigment (*brown induration*). Both induration and ordinary œdema affect especially the bases of the lower lobes. As a result of local interference with the circulation, some transudation of fluid into the pleural cavity (hydrothorax) often occurs, and there is more or less proneness to inflammatory lesions of the lung, either in the form of bronchitis, pneumonia, or pleurisy.

Liver.—The hepatic vein opens into the inferior vena cava so close to the right ventricle that the influence of cardiac disease upon the circulation of the liver can be readily understood. The organ enlarges considerably, and becomes darker in colour, and in advanced conditions acquires a peculiar appearance of red, yellow, and white mottling, to which the name of *nutmeg liver* has been applied. On section the centre of each nodule is seen to be occupied by the enlarged hepatic vein-rootlet transversely divided, and the adjacent central zone of the lobule is dark-red or purple; outside this is a zone of yellow colour, from the retention of bile within it; and outside this the most external zone of the lobule is of white or grey colour, which the microscope shows to consist of cells in a state of advanced fatty degeneration.

Kidneys.—These are, as a rule, simply congested, becoming in consequence larger and dark-coloured; but from long-continued congestion a certain amount of fibrous tissue may develop, and by its irregular distribution and contraction may produce a granular condition of the surface.

Other Abdominal Organs.—The *spleen* becomes hard and darker than normal, and, though varying in size, is often smaller. The congestion of the *stomach* and *intestine*, like that of the spleen, is, of course, secondary to that of the liver, since the veins derived from these organs empty themselves into the portal vein. The mucous membrane becomes congested, and after death considerable distension of the vessels, and sometimes hæmorrhages into the substance of the mucous membrane, may be seen. *Ascites*, or dropsy of the peritoneal cavity, is another result of the obstruction to the circulation in the radicles of the portal vein.

Since malignant or ulcerative endocarditis is frequently grafted upon chronic valvular disease, the different organs may be the seat of embolic *infarcts* (*see* pp. 502, 505). The lungs, moreover, may contain infarcts in the later stages of heart disease, independent of the acute form of endocarditis. They occur as wedge-shaped or conical blood-coloured masses, occupying commonly the lower lobes, and especially their lower edges, and appear at first sight as if they were simply hæmorrhages into the substance of the lung. Accordingly they have been described as *pulmonary hæmorrhage;* but in their shape, distribution, and subsequent progress they resemble precisely the embolic infarcts which occur in the spleen and kidneys, and no doubt the hæmorrhage is determined by the impaction in the arteries of fibrinous masses formed from the blood in the recesses of a dilated right ventricle or right auricle. Commonly about an inch in diameter, they may sometimes reach a large size (*see* Embolism).

Physical Signs.—Amongst the clinical features of valvular disease, we may first consider the auscultatory phenomena which accompany it; but it must not be forgotten that, although the most valuable information may be obtained from the alterations in the heart-sounds, no safe estimate for purposes of prognosis or treatment can be made without a thorough investigation into the size and action of the heart, and into remoter evidences of the way in which the circulation is carried on.

It has been already stated (p. 493) that valvular disease leads to a modification of the natural sounds of the heart, so that more prolonged sounds are heard, called *murmurs* or *bruits;* that such murmurs may occur during the contraction of the ventricles (*systolic*), or during their dilatation (*diastolic*); and that they arise in consequence of *obstruction*, or *stenosis*, at the valvular orifices hindering the flow of blood in its natural course, or from imperfection, or *incompetence*, of the valves, which allows the blood to flow back, or *regurgitate.*

If, now, we consider that each of the four cardiac orifices is provided with a valvular apparatus which may be diseased, we shall see that we can have at each of them a murmur due to *obstruction* (or *stenosis*), and a murmur due to *regurgitation* (or *incompetence*). It will be further observed that at certain orifices the obstructive murmurs are systolic, and the regurgitant are diastolic; while at the other orifices the regurgitant murmurs are systolic, and the obstructive are diastolic.

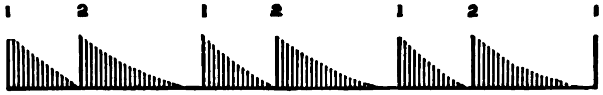
Thus, an obstruction at the aortic orifice gives rise to a murmur during the contraction of the ventricle, which forces blood past the obstruction; hence it is a *systolic* murmur (Fig. 23, between 1 and 2).

Incompetence of the aortic valves gives rise to a murmur during the dilatation of the ventricle, because when the ventricle ceases to contract, the aorta recoils upon the column of blood within it, and forces it against, and partly through, the now incompetent sigmoid valves; hence there is a *diastolic* murmur (Fig. 23, between 2 and 1).

Incompetence of the mitral valve causes a murmur during the contraction of the ventricle, because it is during systole that these valves are called into play; hence with their failure a *systolic* murmur results (Figs. 25 and 26, between 1 and 2).

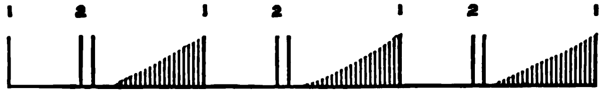
Obstruction at the mitral valve causes a murmur during the dilatation of the ventricle, because during this dilatation only can a current of blood pass from the auricle into the ventricle. Hence, such a murmur must be *diastolic* in the sense that it takes place during ventricular dilatation. But it may be early, mid, or late. Frequently the murmur of mitral obstruction is late diastolic or *presystolic* (Fig. 24, between 2 and 1). This appears to result from the manner in which the auricle contracts. In the normal heart when the ventricle contracts the mitral valves close, and the auricle begins to dilate as blood pours into it from the pulmonary veins; but when the ventricle begins to dilate, the auricle does not thereupon contract, but both cavities are for a time in a state of dilatation; and this is in the first half of *diastole*. Then the auricle, being completely filled, contracts during the latter part of diastole, and its contraction is immediately followed by that of the ventricle. If an obstruction exists at the mitral orifice, it will be especially during the *auricular* contraction that vibration and sound will be produced by the greater force of the current at that time; and hence the murmur produced will occur just before ventricular systole, and will be *presystolic*. Because it is believed

FIG. 23.



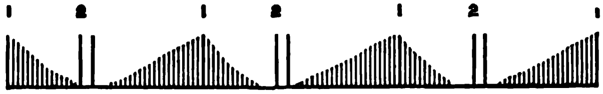
Aortic Disease. Systolic and Diastolic Murmurs.

FIG. 24.



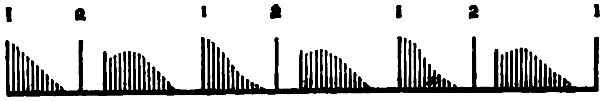
Mitral Disease. Presystolic (late Diastolic) Murmur, with Reduplicated Second Sound.

FIG. 25.



Mitral Disease. Presystolic and Systolic Murmurs with Reduplicated Second Sound.

FIG. 26.



Mitral Disease. Systolic and Mid-Diastolic Murmurs, with Reduplicated Second Sound.

In the above diagrams Nos. 1 and 2 represent the first and second sounds respectively, and the shaded lines between them show the periods of time occupied by different murmurs.

to occur during the contraction of the auricle, it has also been called *auricular systolic.* This murmur is further characterised by a peculiar rough, churning, or rumbling quality, and by its becoming louder and louder, until it terminates in a very loud first sound. It may be then followed by a systolic murmur, and the combination is represented diagrammatically in Fig. 24.

A *mid-diastolic* murmur is one which begins just after the second sound, and ends appreciably before the next first sound. Often there is a long interval between it and the first sound; sometimes the interval is very short, especially if the heart is beating quickly, but the murmur even then does not get louder towards the end, like the presystolic. It is either blowing or rumbling in character (Fig. 26, between 2 and 1), and is almost invariably accompanied by a systolic murmur of regurgitation. There is thus a combination of a short systolic murmur, a double second sound, and then a long mid-diastolic murmur not running up to the succeeding first sound, resulting in a triple sound which may be imitated by the words "feu-tĕ-feu."

An *early diastolic* is the least frequent of the murmurs of mitral obstruction; it is blowing in character, generally of short duration, and rarely heard alone. Its position in the cardiac rhythm is that of an aortic regurgitant murmur, and may be represented by Fig. 23, between 2 and 1.

The explanation of the early and mid-diastolic murmurs of mitral obstruction is not so simple as that of the late, or presystolic. The former may take place during the active dilatation of the ventricle: the latter during the passive flow of the blood from the auricle to the ventricle. There is no difficulty in believing this, since the passive flow of blood in the jugular vein is competent to produce a very loud sound, the *bruit de diable*, or venous hum; but it might be thought that if the passive flow of blood produced a murmur in the middle of diastole, the contraction of the auricle ought in the same heart to produce a well-marked presystolic immediately continuous with it. The absence of such a continuation into the presystolic time may, perhaps, be explained by the fact that in such cases the auricle is generally much dilated and weakened.

What is here stated of the aortic and mitral valves may be said, *mutatis mutandis*, of the pulmonary and tricuspid valves.

The relation of the murmurs to the rhythm of the heart and the flow of blood through it may be tabulated as follows:

Orifice.	Lesion.	Murmur.	
Aortic or Pulmonary.	Obstruction.	Systolic.	
	Regurgitation.	Diastolic.	
Mitral or Tricuspid.	Obstruction.	Diastolic.	Early. Mid. Late (Presystolic).
	Regurgitation.	Systolic.	

Of these, the pulmonary regurgitant and tricuspid obstructive murmurs are very rare; and murmurs actually due to pulmonary obstruction are less frequent than the remaining five, although a systolic murmur over the region of the pulmonary artery is quite common in association with changes in the quality or quantity of the blood, and is known as a hæmic or functional murmur (*see* Anæmia).

Obviously, the eight possible lesions above indicated (obstruction and regurgitation at each of the four orifices) cannot be distinguished solely by the relation of their murmurs to the sounds of the heart. But we find the means of discrimination in the different points of the præcordial area at which the several murmurs are best heard; and these are determined not so much by the actual position of the valve below the surface as by the direction of the current of blood which is flowing past the orifice at the time, and in which, indeed, the sound-vibrations are largely or entirely produced. Indeed, three of the orifices (aortic, mitral, and tricuspid) lie so close together that if the murmurs were heard only at the orifice concerned, it would be very difficult to distinguish the different kinds. But the flow of blood in the aorta from midsternum towards the right clavicle, in the pulmonary artery from the sternum upwards towards the left, and in the heart from auricle to ventricle, conveys each murmur along a special path; and the reflux of blood through the aortic valves into the ventricle, and through the mitral valves into the auricle acts in a similar way in the case of regurgitant murmurs. The term *area* (mitral area, aortic area) is often applied to the part of the præcordia or adjacent chest-wall, where a particular murmur is commonly heard, and in auscultating the heart for valvular disease these areas must be successively examined.

Aortic obstructive murmurs are heard with greatest intensity at the junction of the third right costal cartilage with the sternum, and at the extremity of the second right intercostal space; they can be traced upwards towards the inner half of the right clavicle, and into the vessels of the neck.

Aortic regurgitant murmurs are heard with greatest intensity over the sternum, at the level of the third costal cartilages, and are traceable down the sternum, often to the base of the ensiform cartilage; sometimes also downwards to the left, in the direction of the apex of the heart.

When these two murmurs are combined they may be heard together over the junction of the right third costal cartilage with the sternum; sometimes they are only heard separately, and then respectively above and below this point.

Mitral obstructive murmurs are heard most loudly at the point of impulse of the heart against the chest; though sometimes audible more or less imperfectly between this point and the sternum, they are always best heard at this spot, and are often strictly limited to an area of an inch or an inch and a half in diameter. The stethoscope should always be placed over the actual heart-beat, as found by examination, and not only over the spot where the apex should be normally found.

Mitral regurgitant murmurs are mostly heard with greatest intensity at the apex of the heart, but they are commonly widely diffused, slightly over the præcordial region, towards the sternum and the base of the heart, and also outwards to the left. In the axilla they often lose in loudness, but are again heard at the angle of the left scapula, and even all over the base of the left chest, and over the base of the right chest; sometimes, at least on the left side, as loudly as in front.

Pulmonary obstructive murmurs are heard with great intensity in the second left intercostal space at its inner end, and can be traced outwards in that space, and upwards towards the left clavicle.

Pulmonary regurgitant murmurs are heard at the junction of the third left costal cartilage with the sternum, and thence downwards over the right ventricle, along the left border of the sternum.

Tricuspid obstructive murmurs have been heard, with a presystolic or mid-diastolic rhythm (like mitral obstructive murmurs) at the left side of the sternum, over its junction with the fourth costal cartilage.

Tricuspid regurgitant murmurs are heard at the lower half of the sternum, over an area corresponding pretty closely to the part of the heart left exposed between the two lungs, but they are sometimes limited to the base of the ensiform appendix, sometimes extend to the right nipple, and are probably heard at the apex when dilatation carries the right ventricle in that direction.

In applying the above rules to any cases before us we must bear in mind the following facts:—(1) Valvular disease may exist without a murmur being produced. (2) Very loud murmurs are heard over a large extent, and so encroach on the areas of healthy valves. (3) Two or more valves may be diseased at the same time, as, for instance, the aortic and mitral together; the mitral and tricuspid together; or even the aortic, mitral, and tricuspid at the same time. (4) Besides valvular disease many other conditions (aneurysm, anæmia, and certain diseases of the lungs and pleura) give rise to murmurs over the præcordia.

In the presence of these valvular murmurs, the natural heart-sounds may be considerably altered or partly disappear. Since the second sound of the heart is due to the closure and vibration of the sigmoid valves, disease of these valves resulting in their imperfect closure must be followed by a diminution in the loudness, or by complete abolition of the second sound corresponding to the diseased orifice, so that with a regurgitant aortic murmur the second sound is enfeebled or absent. The same applies to the mitral valve; its perfect closure contributes to the first sound, which must be weakened when regurgitation is present. Since both sides of the heart contribute to the natural sounds, the diseases of one side alone may fail to abolish them. Another frequent modification in heart disease is accentuation of the second sound at the pulmonary orifice; this occurs especially in mitral regurgitation, and is brought about by the increased pressure in the left auricle, pulmonary veins, and pulmonary circulation generally. Mitral constriction in early stages is accompanied by an accentuated first sound at the apex, and a reduplication of the second sound at the base, or lower sternum.

Thrills.—The thrill, or *frémissement cataire*, is only an occasional physical sign of valvular lesion. It is never present without a murmur, and is, indeed, due to the fact that some of the vibrations which cause the sound are of a nature to be felt also. It is most common in mitral constriction, and accompanies a large proportion of presystolic (or late diastolic) murmurs, and some mid-diastolic murmurs. Thrills with other murmurs are comparatively rare, and the valvular lesions which they accompany may be arranged in the following order of frequency: pulmonary stenosis (congenital), tricuspid regurgitation, aortic stenosis, aortic regurgitation, mitral regurgitation, tricuspid stenosis.

The other physical signs of valvular disease are due to the changes in the size and shape of the heart—namely, hypertrophy and dilatation. These are dealt with in detail in another place.

Symptoms.—In describing these, it will be best at first to limit ourselves to disease of the valves on the left side, the aortic and mitral valves. Pulmonary valve disease is rare except in the congenital form, which will be considered hereafter, and tricuspid disease is mostly secondary to mitral affections.

Disease of the aortic and mitral orifices may exist practically without any symptom for long periods, and may be detected only on auscultation. This is explained either by the slightness of the lesion or by the hypertrophy of the muscular walls being sufficient to meet the additional strain upon them, so that the valvular lesion is *compensated.* But where the compensation is insufficient, or where hypertrophy is accompanied by dilatation, symptoms sooner or later manifest themselves.

Mitral disease may be taken first, as being the most frequent. The symptoms are in many points the same, whether the disease be obstructive or regurgitant. The early symptoms are pain or distress at the heart, palpitation, shortness of breath, and swelling of the feet; in later stages, evidences of failing circulation in the various organs of the body. Congestion of the lungs is shown by cough, by mucous expectoration, by occasional hæmoptysis, which may arise from the pulmonary infarcts above described, by orthopnœa at night or continually, and by dyspnœa on the slightest exertion. On examination crepitations will be heard at the bases of the lungs, and in advanced cases dulness, with deficient entry of air, and deficient tactile vibration. General venous stagnation is shown by a rich red colour or actual lividity of the lips, cheeks, ears, and extremities, and by the occurrence of anasarca. The congested liver is large and smooth, reaching perhaps in the abdomen to the umbilicus; and the skin is slightly jaundiced, the yellow tinge of the forehead combining with the deep red of the lips and cheeks to give a very characteristic appearance to the sufferer. Other results of the hepatic stagnation are ascites, enlargement of the spleen, and more or less frequent vomiting. The secretion of the kidneys is also affected, the urine being scanty from low arterial pressure, reduced perhaps to ten or fifteen ounces daily, high-coloured, depositing large amounts of urates and containing albumen and fibrinous casts. Drowsiness or restlessness, and in advanced cases occasionally delirium, show the effect upon the circulation of the brain. Death takes place ultimately from cardiac failure, from œdema of the lungs, from sloughing of the skin and exhaustion, or from some other complication.

The differences between mitral regurgitation and constriction are mainly seen in their effects upon the heart and the vessels, and in the average time they take to develop symptoms.

Mitral Regurgitation.—The left ventricle becomes dilated and hypertrophied; the physical signs are the displacement of the impulse downwards and outwards, the systolic murmur audible at the apex, in the axilla, and at one or both bases behind, and the accentuation of the second sound over the pulmonary artery. In early stages the impulse is more or less heaving and regular, the pulse soft, with a high percussion wave, a rapid fall, a feebly developed tidal wave, and moderate dicrotism. In later stages the heart becomes irregular, with a more and more feeble impulse, while the pulse is correspondingly small, feeble, irregular, and compressible, and its sphygmographic tracing shows a respiratory wave (p. 500).

Mitral constriction at first affects rather the left auricle than the left ventricle. The former is hypertrophied in its endeavours to overcome the obstruction, and the latter remains of its normal size. Thus in early stages the impulse may be in its usual situation, and the condition is revealed only by the characteristic presystolic murmur, audible chiefly at the impulse, rough, rumbling, or churning in character, and running quite up to the first sound, which is generally loudly accentuated, while the second sound is doubled. Where the murmur is best heard there is often a palpable vibration, or thrill, which is, like the murmur, presystolic in rhythm.

In later stages mitral constriction is apt to be complicated by either tricuspid or mitral regurgitation; the presystolic murmur is less often observed, being replaced by the combination previously described (*see* p. 515). Sometimes this and the presystolic alternate on succeeding days in the same case. Exceptionally, the murmur may be early diastolic. Sometimes mitral constriction is unaccompanied by any murmur at all; and not infrequently, there is no diastolic murmur, but a reduplicated second sound and a short systolic murmur, inaudible behind, of which it is not always easy to say whether it is a murmur of mitral regurgitation or not.

In the latest stages of cases of mitral constriction the heart's action is most irregular and tumultuous. The rhythm of the diastolic sound is incapable of recognition, and there seems to be a continuous rumble or roaring sound with a systolic murmur interposed at intervals.

The pulse in early stages may be absolutely normal, of medium pressure, and the heart regular; but in later stages irregularity is the never-failing characteristic, and it often happens that at every fourth or fifth beat the pulse expands twice close together—*i.e.*, it receives a second wave of blood from the heart before the first has been passed over to the arterioles. Tracings of pulses in mitral disease are shown in Fig. 27.

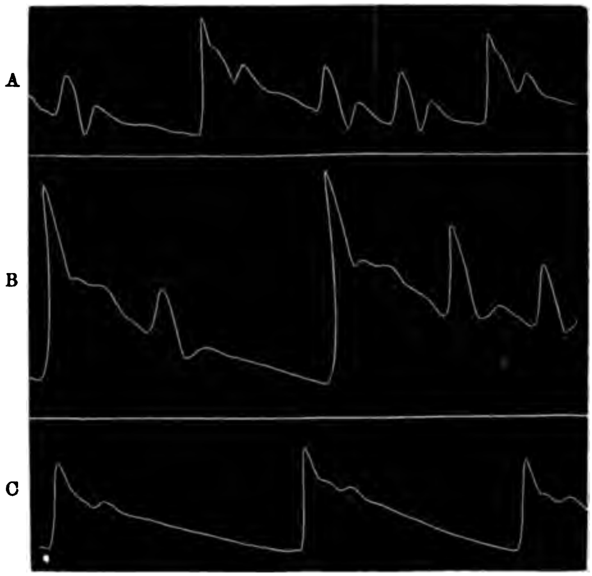
Mitral constriction often gives rise to hæmoptysis quite early, and is perhaps more often than mitral regurgitation the cause of hemiplegia from embolism of the cerebral arteries. As to their relative duration and fatality it is difficult to speak, because while the majority of cases of regurgitation arise out of rheumatic fever, many cases of constriction give no previous history by which the onset can be fixed; and further, either one may be secondary to the other. Mitral regurgitation is sometimes more rapidly fatal than ever constriction can be, terminating within a few months of the rheumatism which has caused it. On the other hand, some cases of regurgitation last very many years. Mitral constriction seems to hold an intermediate position.

Obstruction and regurgitation are often combined at the mitral orifice; they give rise to various combinations of systolic and diastolic murmurs, of which some have been already mentioned (*see also* Figs. 24–26).

Aortic Disease.—The most common form of aortic disease is that in which evidence of both obstruction and regurgitation is present; but the symptoms are mainly due to regurgitation. Next most common is regurgitation alone, and pure aortic obstruction is relatively rare. Aortic valve disease arises much less frequently than mitral valve disease from rheumatic fever, and is often the result of continued strain upon the circulation, especially from the excessive use of the arms, such as arises in blacksmiths, sawyers, and others with laborious occupations. Sudden rupture of the valves also sometimes takes place.

Regurgitant cases are characterised by the murmur already described (*see* pp. 513, 516). The heart is commonly dilated and hypertrophied, and the impulse is carried downwards, and slightly outwards. The effect on the pulse is peculiar and characteristic; the hypertrophied ventricle drives the blood with great force into the arteries, causing a high percussion wave, but, the valves closing imperfectly, the column of blood is not sustained, and the dicrotic wave is badly developed from the slightness of the recoil (Fig. 28). The sudden rise of the pulse-wave, and its equally sudden subsidence, yield a peculiar sensation to the finger, which is expressed by the various names given to this form of pulse, such as *kicking*, *splashing*, *water-hammer*, and *shotty*. The terms *refluent* and *collapsing* refer to the sudden subsidence of the wave, which is, indeed, its essential feature. All over the body the sudden and extensive movements of expansion and contraction in the arteries produce marked effects. The vessels of the neck throb visibly and often painfully; the digital arteries can be felt with unusual dis-

FIG. 27.

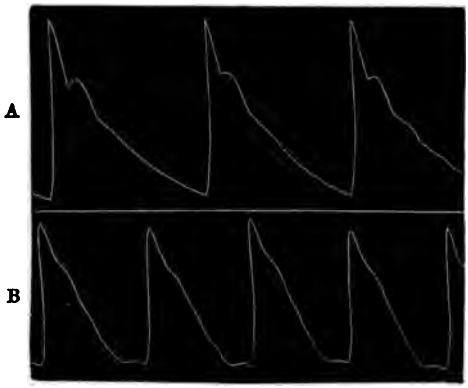


A. Irregular Heart of Mitral Regurgitation, complicated by Renal Disease. Pressure, Six Ounces.
B. Irregular Heart of Mitral Constriction. Pressure, Three Ounces.
C. Pulse of Mitral Constriction under Treatment. Pressure, One and a Half Ounces.

tinctness, and the pulsation of the retinal arteries can be easily seen with the ophthalmoscope. Aortic incompetence may also cause *capillary pulsation.* This may be seen under the nails, in the cheeks, or in the area of dilated capillaries produced by drawing a sharp point over the surface of the forehead; or by pressing a piece of glass (*e.g.*, a microscope glass-slide) upon the mucous membrane of the inverted lower lip. In either case the vascular area under observation becomes alternately darker and paler with each beat of the heart.

Patients with aortic regurgitation are often markedly *anæmic*, with pale face and lips, and mucous membranes; but when, in the course of time, secondary mitral regurgitation takes place, the venous system stagnates, and congestion of the lips and cheeks replaces the former bloodlessness. Shortness of breath, cough, and mucous expectoration, swelling of the feet, pain at the upper part of the sternum, especially on exertion, scanty urine, and albuminuria, are the common accompaniments of this disease, and a termination by sudden syncope is much more frequent than in other forms of valvular disorder.

FIG. 28.



A. Pulse of Aortic Regurgitation. Pressure, Three Ounces.
B. Pulse of Aortic Regurgitation. Pressure, Four and a Half Ounces.

In pure *aortic stenosis* the murmur is simply systolic, audible in the second right intercostal space near the sternum, and traceable up towards the right clavicle. If stenosis is considerable a thrill may be present, felt at the same spot, and also systolic. The heart will be hypertrophied or dilated, proportionate to the amount and duration of the obstruction. The pulse is often characteristic; the obstacle interposed in the current of blood prevents the full effect of the ventricular contraction upon the column of blood in the systemic arteries, and the pulse can be felt to have lost its suddenness and to rise quite slowly. The pulse tracing is then *anacrotic*, that is, the percussion-wave is lower than the succeeding tidal wave, and appears as an elevation on the ascending limb. In the extreme variety of this form this wave is rounded off, or entirely absent, and the tracing resembles that shown in Fig. 29, A (p. 562). In some cases the pulse has a double beat, and the tracing shows both percussion and tidal waves very high, with a marked depression between them (*pulsus bisferiens*).

The symptoms of combined aortic obstruction and regurgitation, or double aortic disease as it is often called, are mainly those due to regurgitation. A systolic and diastolic murmur are heard, forming the *to-and-fro* murmur, and the patient is commonly anæmic in the early stages; later on, the left ventricle becomes more dilated, the mitral orifice widens, the valve becomes insufficient, and the symptoms due to this condition show themselves.

Tricuspid Regurgitation.—On the right side of the heart, the only form of valve disease that is at all common is *tricuspid incompetence* leading to regurgitation; and this is mostly due to distension of the right ventricle, causing enlargement of the tricuspid orifice, which the cusps of the valve are unable to cover; and only rarely to primary disease of the valve itself. Such dilatation of the right ventricle may arise from any increased pressure in the ventricle and pulmonary artery, whether from chronic disease of the lungs (emphysema, bronchiectasis, very chronic phthisis) or from incompetence or obstruction at the mitral valve. It is thus frequently associated with mitral disease, and the other forms of left-side failure. It is commonly accompanied by the evidence of dilatation of the right heart, and by the various degrees of œdema, anasarca, and venous congestion which indicate a difficulty in the return of blood to the right heart and lungs. These have already been enumerated under the later symptoms of mitral disease. The signs more characteristic of tricuspid regurgitation are, the systolic murmur, and the jugular and hepatic venous pulsation already described (pp. 517, 500). A systolic thrill over the lower end of the sternum occasionally accompanies the murmur.

Tricuspid obstruction (*see* p. 517) is less common, and is generally observed in conjunction with other valve disease. No special group of symptoms can be referred to it apart from those seen in tricuspid regurgitation.

Disease of the *pulmonary valves* is mostly congenital if chronic; and if acute, it is the result of malignant endocarditis. In the former, the valves are often united together in a cone, and cause much *obstruction*, though they may close perfectly. The murmur is systolic (*see* pp. 515, 517), and is often accompanied by a marked systolic thrill over the same area. The obstruction leads to dilatation of the right ventricle, and at the same time so far hinders access to the lungs, that a very imperfectly aërated blood circulates, and the patient is habitually cyanosed (*see* Congenital Malformations).

Pulmonary *regurgitation* sometimes occurs as a result of mitral stenosis, the valves yielding before the great pressure in the pulmonary artery; the murmur is heard down the left side of the sternum.

Malignant endocarditis may cause a double (systolic and diastolic) murmur at the pulmonary orifice like that of aortic disease, the respective murmurs having the position previously noted. The symptoms under such conditions have already been detailed (*see* Malignant Endocarditis).

Complications.—Every result of simple venous stagnation must be considered a part of heart disease, such as red and brown induration of the lung and albuminuria. But pleurisy, pneumonia, embolism, and malignant endocarditis may be regarded rather as complications. With a failing circulation fibrin may be deposited upon the valves and in recesses of the dilated cardiac cavities. Hence detached particles may be carried into the cerebral vessels, causing hemiplegia; into the splenic or renal vessels, producing the characteristic infarcts, or more rarely into the vessels of the extremities, leading perhaps to gangrene. Malignant endocarditis is frequently grafted upon chronic disease.

Diagnosis.—This is generally by no means difficult, and rests mainly on the direct evidence obtainable from the heart's impulse and sounds. Here it must not be forgotten how much information as to the heart's position, action, and valvular incompetence can be got by the eye and the hand; and these should always be used in conjunction with the stethoscope. The murmurs of valve disease are apt to be confounded with those due to other conditions.

Anæmia produces a harsh systolic murmur over the pulmonary area. Seeing the rarity of organic pulmonary disease, this is mostly distinctive enough, but with very considerable anæmia murmurs extend over the whole præcordial area, arising, no doubt, at other orifices besides the pulmonary. As in such cases the patients are short of breath, with tendency to palpitation, and to swelling of the feet, the diagnosis may be difficult. The marked pallor of the anæmic cases, the absence of history of rheumatism, or other precursor of heart disease, and the diminution of the murmur under the use of ferruginous tonics, are points which will help. But anæmia may also undoubtedly itself be a cause of mitral regurgitation; the deficient quality of the blood causes malnutrition of the wall of the ventricle; this dilates, the mitral orifice yields, and regurgitation is the result. This is, indeed, an actual lesion, and the murmur is immediately due to structural changes; but inasmuch as they are primarily due to a condition of the blood, which, together with its results, is curable, this murmur is often spoken of as *functional* or as *hæmic*. The diagnosis must, at any rate, be made between this and chronic valvular disease, and it can generally be effected by a consideration of the preceding and associated circumstances—viz., the absence of rheumatism and the decided anæmia.

Aneurysm of the aorta frequently gives rise to a murmur at the base of the heart which may be mistaken for that of aortic obstruction. Indeed, a simple systolic murmur in the aortic area, unaccompanied by regurgitant murmurs, is much more often due to aneurysm than to stenosis of the valves. Abnormal pulsation to the right of the sternum, and increased area of dulness, should be sought for as further evidence. If the murmur is localised at a point not strictly corresponding to the known areas of valve disease, aneurysm is still more probable.

Pericarditis often gives rise to a to-and-fro murmur, very like that of double aortic disease. It is, however, usually rougher, less uniform in loudness over a large area, not strictly localised to the usual area of aortic disease, and perhaps here and there not strictly synchronous with the two periods of the heart's beat. A short history of acute illness, unusual pain or distress at the heart, increased area of præcordial dulness in an upward direction, and absence of splashing pulse, point to pericarditis.

Another difficulty arises from sounds, synchronous with the heart's action, produced outside the heart—*exocardial* murmurs, of which the *cardio-pulmonary* forms are more frequent. The most common of these is a short, high-pitched, systolic murmur, often limited to the apex, which is heard in persons of excitable temperament when they are under medical examination. The heart beats quickly and very violently, and the murmur may be heard at the left scapula behind as well as in front. Others are produced by displacement of the heart, as when it is compressed by pleural effusion, or by deformities of the thorax; and others by morbid conditions of the lung and pleura immediately adjacent to the heart, and mostly on the left side. Very extraordinary murmurs are sometimes heard when a large pulmonary cavity is in close contact with the heart, the air being driven suddenly out of the cavity with each cardiac impulse.

In later stages the diagnosis of heart disease presents fewer difficulties. There is often in children and young people a superficial resemblance between mitral disease and *phthisis*; the former may produce pallor, emaciation, and hæmoptysis; but a careful examination will at once show whether heart or lung is primarily at fault. It is sometimes a hard task, in old people, to distinguish cases primarily cardiac from those which are primarily *renal*. *Chronic renal disease* may bring about hypertrophy of the heart, and even dilatation and murmur, and the case will then closely resemble one of mitral disease with secondary albuminuria. The difficulty is further increased by the fact that kidneys in a state of chronic congestion from heart disease may become granular, and that a heart dilated, as a result of extreme arterial tension in renal disease, will cause secondary stagnation in the venous system, like one affected with primary mitral disease. In primary heart disease one must look for the history of rheumatism or other cause of endocarditis. The urine is commonly high-coloured, depositing urates, with an amount of albumen which varies according to the efficiency of the heart as influenced by treatment; and the pulse is small and of low tension. But in renal disease the urine is more likely to be pale, though also scanty, to have a more uniform quantity of albumen, and the pulse has the characteristic high tension.

It still remains to say something of the diagnosis of the different forms of valvular disease one from another. This depends for the most part on the character of the murmurs, and the extent to which they are audible over the præcordial area. A murmur may be conveyed beyond the area of one valve into the area of another, when it will be necessary to compare carefully the intensity of the sound at different points. Aortic regurgitation and mitral regurgitation are nearly always indicated by their characteristic murmurs; though it is doubtful if one can speak positively as to mitral regurgitation unless the murmur is heard behind at the angle of the scapula as well as in front at the apex, murmurs of more limited range being possibly produced within the ventricle itself. On the other hand, mitral obstruction frequently exists without its characteristic murmur, which may require for its development a certain force of auricular contraction in proportion to the obstruction at the valve. Pure presystolic murmurs, pure diastolic murmurs, and others intermediate in rhythm, when heard at the exact impulse (and not heard at the base), are very strong evidence of mitral obstruction. But murmurs almost identical with these are sometimes produced at the apex by aortic regurgitation, a fact first noted by Austin Flint.

In cases of incompetent mitral and tricuspid valves, with resulting regurgitation, it has also to be determined whether the lesion is a chronic, old-established contraction of the valves, or an acute disease of the valves, or a dilatation of the orifice, whether recent or old. It has already been shown that the ventricles may become dilated from alterations in the nutrition of their muscular walls, and this dilatation has been shown to prevent the normal tension of the auriculo-ventricular ring bounding the orifice.

Anæmia, chorea, and pyrexial disorders are the causes of this kind of incompetence.

Prognosis.—The efficiency of the heart as a propelling organ, and not the loudness of the murmur, must be taken as the guide to an estimate of the duration of life. Acute temporary dilatations may be entirely recovered from, but pronounced valvular disease is practically incurable. The most that can be done is to preserve the efficiency of the organ as long as possible, and this may be to the end of a long life. The object is attained if the lesion is but slight, or if, when the patient is relieved from undue strain of body or mind, and is put under the best possible conditions in every respect, an adequate compensatory hypertrophy is developed. Even under relatively adverse circumstances, with a comparatively severe lesion, the heart may remain *in statu quo* for years. In any given case one should take into consideration the valve affected, the condition of the heart at the time, and the history of the patient's illness. When a patient comes under treatment for the first time, his temporary improvement is highly probable; but frequent relapses argue insufficient compensation, and a short future. The amount of hypertrophy and dilatation compared with the duration of the illness, so far as it can be estimated, will show the probable rate of progress that may be expected; but considerable allowance must be made for the effects of treatment, if it has not been thoroughly instituted. As to the valve affected, something has been already said. Aortic obstruction is least serious, aortic regurgitation most.. Mitral obstruction often remains stationary for many years, but much constriction may be very rapidly fatal. Free mitral regurgitation also tends to shorten life considerably. The prognosis is naturally more grave when two or more lesions co-exist; aortic disease rapidly terminates when the mitral valve becomes secondarily involved, and tricuspid regurgitation is a serious complication of left-side disease. Persisting pyrexia should make one suspect malignant endocarditis, when the prognosis becomes at once unfavourable.

Treatment.—The mere presence of an endocardial murmur, the result of old disease, does not call for any special treatment by drugs so long as the ventricle remains unaffected, or slightly hypertrophied, and not dilated. All that is necessary is to warn the patient against over-strain and over-exertion, to maintain the nutritive powers of the body, without allowing excess, and in case there is a tendency to deficient nutrition to meet it by cod-liver oil, iron, and other tonics. When symptoms definitely show themselves, such as shortness of breath and palpitation, more decided action is required; and the treatment involves three indications which may have to be pursued with more or less vigour, according to the stage or severity of the disease :—

infusion or 10 or 15 minims of the tincture may be given every three or four hours at first, and after twelve or twenty-four hours less frequently or in smaller doses. The usual effect is that the heart beats more slowly and powerfully, free diuresis takes place, dropsy and dyspnœa diminish, and the comfort of the patient is secured. But its action requires to be watched: an overdose causes very slow pulse, with sickness and headache.

Digitalis is not suitable for all conditions of valvular disease; it is contra-indicated in cases of hypertrophy without dilatation, when the heart's beat is powerful, of moderate quickness only, or even slow, and nearly or quite regular. It only increases the violence of contraction in such conditions, which arise more often in connection with aortic disease than with mitral disease, and hence digitalis is less often of use in the former.

In aortic regurgitation the fact that digitalis prolongs the diastole seems in one respect to be a disadvantage: for since during the diastole the blood flows back from the aorta into the ventricle, the longer the diastole the greater the regurgitation, and the less for the time being is the supply of blood to the brain and other tissues. There would thus be a greater risk of syncope from aortic disease during the use of digitalis; but the tendency is probably as a rule neutralised by the more powerful action of the heart during systole. As a fact, digitalis has generally a smaller field for service in the slower and more forcible action of aortic disease; but when the ventricle is acting feebly and irregularly, it may be used.

Some other drugs have a similar action with digitalis. The most important of these is strophanthus; of this 5 to 7 minims of tincture (1 in 20) should be given as a dose; it is not so generally trustworthy as digitalis. Convallaria (5 to 30 minims of a 1 in 8 tincture) and caffein citrate (5 to 10 grains) are also employed. Strychnine also acts as a valuable cardiac tonic, and may be given in doses of 5 or 7 minims of the liquor internally, or 2 to 5 injected subcutaneously.

The stimulant action of brandy, ether, or ammonium carbonate is required in the more critical stages.

In aortic disease with hypertrophy, one of the most distressing symptoms is the violent action of the heart, and the throbbing of the great vessels in the neck and over the body generally; and this may be much relieved by the use of a small dose of tincture of aconite (1 to 3 minims), by bromides, or by a small dose of morphia.

Other symptoms and complications may have to be treated. Pain over the heart is often severe, and may be relieved by belladonna plasters, by small doses of morphia internally, or by subcutaneous injection in some cases; but this drug must be used with very great caution, and not at all in advanced cases with much cyanosis. Cough may be treated with small doses of expectorants and sedatives; and vomiting by effervescing salines. Little can be done for the secondary affections of the lungs. Slight pleural effusion may be treated by the application of tincture of iodine; larger effusions may be tapped. Induration of the lung is not amenable to local treatment; pulmonary hæmorrhage is rarely sufficient to threaten life, and does not require styptics.

Cerebral embolism, again, is beyond the reach of direct treatment, but embolism of an artery in one of the limbs must be met by the application of warmth, to avoid as far as possible the supervention of gangrene. Throughout, the diet should be in moderate quantity, light, easily digestible, and unstimulating. With a much weakened heart, and in late stages, however, stimulants in the form of brandy, whisky, or sherry, will form an essential part of the treatment.

The hill-climbing of Oertel and the baths with graduated exercises employed by Schott, at Nauheim, do not seem so suitable for the treatment of valvular lesions as for other less permanent lesions of the heart, unless in quite early stages. They will be described later (p. 539).

CONGENITAL MALFORMATIONS.

Malformations of the heart arise from defects in its development, which is normally not complete until the closure of the ductus arteriosus and foramen ovale some days after birth; and their origin can be only understood from a knowledge of the manner in which development probably proceeds. The organ commences as a straight tube, receiving a single vein behind, and giving off a single arterial tube in front; it subsequently becomes bent on itself, and for a time consists of two cavities—an auricle and a ventricle. A septum then arises in the auricle, and later a septum grows up in the single ventricle from apex to base. Further, the aorta and pulmonary artery arise by the growth of a septum in the first-formed single arterial tube; and the final change takes place when, in consequence of the diversion of blood to the lungs after birth, the ductus arteriosus and foramen ovale are both closed.

Arrest of this process in any stage will lead to a congenital malformation. It has occurred so early as to leave the heart with only two cavities—an auricle and a ventricle; or with three cavities—a ventricle and two auricles. But these are very rare cases, and the children mostly live but a short time after birth. A more common malformation is that of a *deficient septum ventriculorum*. According to the stage of development at which the arrest has taken place, the deficiency may be a very large one, or a mere perforation in the upper part; and in this latter case the aperture occupies the uppermost tendinous portion of the septum, which lies between the anterior and the right posterior aortic valves, and is known as the "undefended space." When the deficiency is a large one, the aorta frequently arises from the right ventricle, or from both right and left ventricles, and the foramen ovale and ductus arteriosus may one or both be pervious. The defect of the septum ventriculorum is commonly brought about by *constriction at the outlet of the right ventricle*, due to union of the pulmonary valves, to constriction of the pulmonary artery or its orifice, or to a constriction of the infundibular portion. The increased pressure which this produces in the right ventricle is relieved by the blood flowing through the septum into the left ventricle, and the aperture in the septum is thus kept permanently open. If the constriction of the pulmonary outlet takes place after the septum of the ventricles is complete, either the foramen ovale or the ductus arteriosus must remain pervious.

Constriction or obliteration of the aortic orifice or of one auriculo-ventricular orifice sometimes occurs, and similarly interferes with the course of the circulation and the normal development of the heart; and complete transposition of the aorta and pulmonary artery has also been observed.

The ductus arteriosus and the foramen ovale may remain unclosed without any obvious reason—probably, however, from a temporary obstruction to the circulation at the time of birth; but it must be remembered that more or less patency of the foramen ovale is a very common condition in perfectly healthy individuals, a mere fissure or narrow valvular opening being insufficient of itself to allow of any free passage of the blood from one cavity to the other.

Instead of three sigmoid valves in the aorta or pulmonary artery there may be only two, or there may be four. This change may exist in association with other deformities, but if alone it is less likely to give rise to difficulties at birth than to lay the foundation of disease in later life.

Causation.—Little is known of the cause of arrested development of the heart. When pulmonary stenosis appears to have been the primary lesion, this has been attributed to an intra-uterine rheumatic endocarditis; and the fact that the right side of the heart has been affected, rather than the left, which is more susceptible in extra-uterine life, is supposed to be explained by the proportionately greater amount of work performed by the right ventricle during intra-uterine existence.

Symptoms.—Malformations of the heart are commonly accompanied by a series of symptoms, the most prominent of which is a strongly-marked lividity, due to imperfect aëration of the blood. Hence the terms *cyanosis* and *morbus cœruleus* have been used to distinguish these cases, though the former has now a more general application to all conditions of lividity, however produced. In the present case, the lividity is most marked in the prominent parts of the face—the cheeks, lips, nose, and ears, and in the fingers and toes. In slighter cases it is only a richer red than natural; in the severest cases it is purple almost to blackness, and any exertion at once increases the distension of the vessels and deepens the colour. The chronic stagnation tends to cause thickening of the parts affected, and the nose and lips are coarse, while the ungual phalanges of the fingers or toes are thickened much beyond the rest of the fingers, or "clubbed." The blood shows in a remarkable degree the great excess of red corpuscles which is met with in all cases of cyanosis; thus the corpuscles have been found to number from 8,000,000 to 9,000,000 per cubic millimeter and the hæmoglobin may reach from 110 to 160 per cent. of the normal. The patient is incapable of much exertion, from the readiness with which dyspnœa supervenes, and is also peculiarly susceptible to cold or exposure, and easily suffers from attacks of catarrhal bronchitis. In later stages œdema of the legs, ascites, enlarged liver, and albuminuria supervene; or the patient may succumb to bronchitis; or tubercular disease of the lung may be the cause of death.

The **Physical Signs** do not always give precise data as to the malformation present. The præcordial region is sometimes prominent and the dulness encroaches upon the sternum in consequence of the dilatation and hypertrophy of the right ventricle. Most commonly a systolic murmur is heard, either over the præcordial region generally, or over the base of the pulmonary artery, since an obstruction of this vessel is so frequently a factor in the disease. The murmur may be heard behind, or it is localised to the front; sometimes it is accompanied by a systolic thrill. But cases of marked cyanosis may occur without any cardiac murmur.

The exact cause of cyanosis has been much debated, and under the circumstances the view was natural that it was due to a mixture in the heart of venous and arterial blood; but, in other conditions, great cyanosis occurs without any mixture of the two currents; and, on the whole, it seems more probable that the chief cause of the blueness is deficient aëration of the blood from obstruction of the pulmonary artery, and the circulation of the blood thus imperfectly aërated. Simple deficiency of the septum ventriculorum may occur without cyanosis, when the blood passes from the left to the right ventricle, producing a murmur, and perhaps a thrill, in the lower part of the præcordial region; and the ductus arteriosus may remain patent without cyanosis, causing sometimes a prolonged murmur running through systole into diastole, and waxing and waning in loudness.

Prognosis.—Congenital malformations are always unfavourable.

Cases of severe defect live but a few hours or days; others of slighter degree survive five, ten, or twenty years; and even persons with very ill-developed organs have occasionally reached middle age. In any given case the prognosis must depend upon the evidences of cardiac efficiency in the history of the patient, rather than upon any opinion as to the nature of the malformation.

Treatment.—This is entirely palliative. The patient must be kept always thoroughly warm, and protected from exposure to cold and from undue exertion. The symptoms of the later stages must be dealt with as in cases of acquired valvular disease.

MYOCARDITIS.

Myocarditis, or inflammation of the muscle of the heart, may be either acute or chronic. *Chronic myocarditis* can only be recognised in its final stage of fibroid change, and is included in the description of fibroid degeneration (p. 536).

Acute myocarditis occurs mostly in connection with pericarditis or endocarditis. In fatal cases of pericarditis, the layer of muscular tissue immediately under the pericardium is sometimes seen to be paler than normal, as if fatty; and in cases of adherent pericardium, bands of fibrous tissue may be found extending from the surface into the substance of the heart. These seem to show that the inflammation which attacks the pericardium affects at the same time the deeper structures, the early stage of the change being one of infiltration with leucocytes. This condition may be recovered from, or there may be fibrous transformation of the effused products. It occurs, like the pericarditis with which it is associated, chiefly in rheumatism. An idiopathic primary form has been described, but it is extremely rare.

A more local inflammation of the heart's muscle results from endocarditis, where ulceration of a valve extends to its base, and then invades the muscle; or where vegetations or semi-detached fragments of an ulcerating valve set up ulceration in adjacent parts of the endocardium by friction or contact, and this involves the myocardium. The effects upon the general circulation may be of the same kind as those of malignant endocarditis.

A third form is suppurative myocarditis, which is chiefly the result of pyæmia. Small abscesses occur in the substance of the heart, mostly in the wall of the left ventricle, and may approach so near to the pericardium as to rupture into its cavity and set up acute pericarditis. This form of myocarditis occurs especially in connection with, and secondary to, acute necrosis of the long bones (*see* Pyæmia, p. 143).

The **Symptoms** of myocarditis are by no means pronounced. When it accompanies pericarditis or ulcerative endocarditis, they may be entirely lost in the symptoms due to these conditions. It probably causes more feebleness of contraction of the heart, with feeble or irregular pulse, and it increases dyspnœa, and the tendency to collapse; but its share in producing these when pericarditis or endocarditis is present will be difficult to recognise. Some enlargement of the præcordial dulness, from yielding of the ventricular walls, may be expected. The symptoms of abscess of the heart are also uncertain, and the diagnosis from physical signs equally obscure.

Prognosis and **Treatment** have little place where diagnosis is so untrustworthy; but the form associated with rheumatic pericarditis is the only one that can be looked upon as curable, and here the treatment pursued for the relief of the accompanying lesion will be applicable; the support of the failing heart being the main indication.

DEGENERATION OF THE MYOCARDIUM.

The muscular wall of the heart is liable to the following forms of degeneration—*Pigmentary*, *fatty*, and *fibroid*.

PIGMENTARY DEGENERATION.

(*Brown Atrophy of the Heart.*)

The heart is smaller than normal, and the muscular fibre, instead of having a full red colour, is of a dull brownish red, and softer and more friable than is natural. Under the microscope the fibrillæ are seen to contain a number of minute yellow granules. It occurs in senile and cachectic conditions, being common in fatal cases of malignant disease of other organs.

FATTY DEGENERATION.

This change in the muscular fibres must be distinguished from the deposit of fat about the heart (*see* p. 538). In the latter the ordinary adipose tissue is deposited beneath the pericardium, and invades the muscular fibre by the growth of fat-cells between and amongst them. In the former, or true fatty degeneration, the muscular fibrillæ themselves are the seat of minute fat granules, which replace the true sarcous elements and rob the muscle of so much of its contractile tissue. This true fatty degeneration occurs in different forms; the muscular wall may be uniformly affected, or the fatty changes may be limited to a small patch, or to the layer underlying the pericardium, as described under myocarditis, or it may consist of streaks and lines on the inner surface of the heart. When the affection is general the heart is of softer consistence, more easily lacerable, of pale pink or buff colour, and often somewhat larger than normal, from yielding of the affected muscular tissue. When the fat is deposited in lines or streaks it gives a characteristic appearance, the lines of pale yellow colour being often arranged upon the darker red muscle, like the markings of a tabby cat. They are seen mostly on the musculi papillares, and nearly always affect the ventricles, the left more often than the right.

Ætiology.—The causes of fatty degeneration of the heart are general and local. It may be the result of a general tendency to degeneration, such as occurs at an advanced age; it is seen constantly in pernicious anæmia, and often in other forms of anæmia, in purpura and scurvy, and in cachectic conditions, such as phthisis and cancer; in poisoning by phosphorus, and by some mineral poisons (lead, antimony, arsenic). In most of the acute febrile diseases the consistency of the heart is sometimes altered, as the result of a finely granular condition of the muscular fibres, which is probably not to be separated from fatty degeneration. This is the case in enteric and typhus fevers, in yellow fever, diphtheria, small-pox, and measles. The most important local condition is obstruction or narrowing of the coronary artery, by which the nutrition of the heart-wall is necessarily impaired. This may arise from atheromatous or syphilitic changes in the vessels, or from thrombosis or embolism. Fatty degeneration also arises from myocarditis, and happens in connection with long-standing valvular disease, or dilatation from other causes.

Symptoms.—These are mainly those of diminished force in the ventricular contraction. The impulse is feeble; the præcordial dulness may be increased if dilatation co-exists, but is often quite natural. The heart's sounds, especially the first, are faint, and the radial pulse is small and feeble. An apex murmur is occasionally present. The rhythm varies; it may be regular or irregular. The beats may be frequent, or, on the other hand, they are slower than normal, falling to twenty or less in the minute. Pain is not generally prominent as a symptom; and pallor, attacks of syncope, and dyspnœa, chiefly on exertion, but sometimes constant, are the more usual effects. The syncopal attacks may be nothing more than transient faintness, or there may be complete unconsciousness. Occasionally they have much resemblance to cerebral attacks, with a sudden fall, coma, stertor, and convulsive twitchings; or they may be like epileptic fits. Recovery from the attack takes place without hemiplegia. Sometimes the dyspnœa has the characters of Cheyne-Stokes' respiration. Slight œdema of the feet may be present, but rarely a well-marked dropsy, and there is almost entire absence of the signs of venous congestion which are seen in valvular disease. In other respects the functions of the body are badly performed, and nutrition is imperfect. The tissues are soft, muscular power is diminished, the appetite is poor, and digestion is bad. Death may happen in one of the syncopal attacks, or quite suddenly, or by a more or less prolonged asthenia. In a certain proportion of cases, rupture of the heart takes place. Localised fatty degeneration, occurring in the course of old disease of the heart, or in pernicious anæmia, may give rise to no obvious symptoms. On the other hand, the acute change in the heart, which takes place in the course of severe pyrexial illnesses, like typhoid fever, can be readily recognised, and its progress watched. The pulse, hitherto rapid, becomes weak and irregular, the cardiac impulse is feeble, the apex-beat is displaced outwards, and the first sound is so faint as to be scarcely audible. Marked pallor, or in later stages lividity of face, indicates the failure of the circulation.

Diagnosis.—Fatty degeneration does not always give sufficiently clear indications for its recognition. The evidences of feeble cardiac action, not apparently due to valvular disease, or ordinary dilatation, together with marked dyspnœa, or the peculiar syncopal attacks in a person of advanced age, are the chief points to note. Degenerative signs elsewhere are of little value, but their absence might be regarded as opposed to the diagnosis.

Prognosis.—Except in the form that occurs in acute illnesses, the condition is probably incurable.

Treatment.—This must consist in avoidance of undue exertion, of making efforts with the breath held, or of mental excitement; in the use of a diet, with more nitrogenous food and less of the fatty, starchy, and saccharine elements; in moderate doses of stimulants, and the exhibition of tonics, such as quinine, arsenic, iron, and strychnine. Digitalis must be given with caution, and only in cases where the beat is frequent and irregular, with evidence of dilatation.

Cardiac failure in enteric fever requires the free employment of stimulants, with ammonia and digitalis in frequent doses.

FIBROID DEGENERATION.

In this form of degeneration, the muscular tissue of the heart is replaced by white fibrous or connective tissue. The change is in most instances partial, and affects especially the left ventricle, so that streaks and patches of a white colour are seen in the muscular substance. In extreme cases, on the other hand, the whole ventricle may be converted into fibrous tissue, but even here some traces of muscular fibre may be found on microscopical examination. Though the co-existence of peri- or endo-carditis will sometimes show that it is inflammatory (*chronic myocarditis, interstitial myocarditis*), its origin in this way cannot always be demonstrated. It may result from malnutrition consequent upon obstruction of the coronary arteries, or upon the chronic stagnation in the coronary veins, which occurs in various pulmonary disorders. It has been ascribed also to alcoholism, to long-continued congestion, and to syphilis (*syphilitic myocarditis*). The heart affected with fibroid disease may be hypertrophied or dilated, or the subject of adherent pericardium; but often the affected part of the heart's wall is thinner than normal, and it may be bulged out into a distinct aneurysm; indeed, fibroid degeneration is the chief cause of aneurysms of the wall of the heart.

The **Symptoms** of fibroid degeneration are not distinctive. In some cases, apparently perfect health has been enjoyed till the patient has suddenly fallen dead; in others, the symptoms have been those of valvular disease. There may be in these instances evidence of dilatation, or the murmur of mitral regurgitation.

The **Treatment** is that of cardiac dilatation or valvular disease.

RUPTURE OF THE HEART.

Apart from injury, this is mostly a consequence of fatty degeneration; in a very small proportion of cases, its cause is abscess, malignant endocarditis, or aneurysm. It occurs, like fatty degeneration, in old people, and not infrequently follows muscular efforts. The left ventricle was the seat of the rupture in three-fourths of the cases on record. The patient is suddenly seized, and dies almost instantaneously, death being preceded only by intense cardiac pain, pallor, and unconsciousness, or a few convulsive twitchings. In rare cases life has lasted some hours, or even days, with pallor, cold sweats, feeble pulse, and sighing respiration. It may then closely resemble the rupture of an aneurysm into the pericardium. Absolute rest, with the head low, maintenance of bodily warmth by external applications, and of the circulation by small quantities of stimulants frequently administered, are clearly the indications for the almost hopeless treatment of such a condition.

ANEURYSM OF THE HEART.

Intimately related to fibroid degeneration are aneurysms of the walls of the heart; indeed, fibroid degeneration is the cause in nearly all cases. The wall of the cavity affected is weakened at one spot by this conversion of its muscular fibre into white fibrous tissues, and dilates under the pressure of the blood into a sac. The left ventricle is their usual seat, and only a few cases are on record of aneurysms of the other three cavities. In two cases out of three they occupy the apex; they form rounded sacs, of which the communication from the ventricle may be of the same size as the sac itself, or very much smaller. The former variety is more frequent when they arise at the apex—that is, the aneurysm is continuous with the cavity of the ventricle; the latter, more sacculated variety, occurs more often at the side or the base of the ventricle. In size they have been compared to nuts, fowls' eggs, or small oranges; a few have been much larger. The walls are generally very thin, and sometimes infiltrated with calcareous matter; they are lined by endocardium, and mostly contain fibrinous coagulum. They have been found at all ages, from twelve upwards; and in males more often than in females.

Symptoms, if present, cannot be distinguished from those of other cardiac and valvular lesions, with which the aneurysm may indeed be associated; but in a considerable proportion of cases death has taken place either suddenly, and probably from syncope, or, as afterwards proved, from rupture of the aneurysm into the pericardium.

Acute aneurysms of the heart may arise from ulcerative endocarditis of the ventricle wall, in the manner mentioned under Endocarditis, and this is a rather frequent cause of aneurysm of the undefended space (p. 531), as well as of the valves (p. 504). Aneurysms of the undefended spaces are, however, sometimes congenital. In either case, the sac is concave to the left ventricle. The condition is not recognisable during life.

FATTY OVER-GROWTH.

In this condition, which must be distinguished from fatty degeneration, the surface of the heart is overlaid by a large quantity of fat, so that the muscular fibres may be entirely concealed from view. The fat, which is simply an overgrowth of adipose tissue beneath the pericardium, encroaches upon the muscular wall, and, pressing upon the muscular fibres, causes them to waste to a certain extent. It occurs for the most part in persons suffering from general excess of fat, or obesity.

Symptoms.—In the more pronounced cases—like those of fatty degeneration proper, the result of enfeebled action of the heart—there are diminished impulse, faint sounds, and weak, small pulse; dyspnœa on exertion, and occasionally attacks of syncope. Death takes place suddenly in some instances. The symptoms may be in part due to accompanying atheroma of the arteries.

Treatment.—The treatment of fatty infiltration of the heart should be conducted much on the same lines as that of *obesity*, with due consideration of the fact that the heart is affected. Excess in eating and drinking should be avoided; alcoholic beverages should be forbidden. The necessary fluids are better taken shortly before or two hours after a meal than with it. The diet should consist chiefly of lean meat, chicken, fish, green vegetables, a little ripe fruit, while starchy, saccharine, and fatty foods should be reduced to a minimum.

The system of treatment conducted by Schott and others at Nauheim is probably more suitable to fatty over-growth of the heart than it is to severe valvular diseases.

It consists partly of immersion in saline baths, partly of regulated exercises of the muscles of the arms, trunk, and legs conducted slowly against resistance on the part of an attendant. The different springs of Nauheim have a temperature of from 60° F. to 95° F., and contain free carbon dioxide, in addition to the salines, of which the most abundant are sodium chloride, and calcium chloride and bicarbonate. An important influence is attributed to the stimulating action upon the skin of the saline ingredients, and the minute particles of carbon dioxide gas. An elaborate series of movements has been devised, comprising flexion and extension at all the big joints, these movements being made by the patient, but gently opposed throughout by the attendant. No movement is repeated at the same sitting, an interval of rest occurs between any two successive movements, and indications of strain or dyspnœa or distress are the signals for ceasing the exercises.

The results of this combined treatment are stated to be slowing and lessened irregularity of the pulse, great diminution of the precordial dulness, and general improvement in the comfort of the patient.

It is allowed that the waters of Nauheim may be successfully imitated by the addition to other waters of equivalent proportions of sodium chloride (1 to 3 per cent.) or calcium chloride (·2 to ·5 per cent.), or sufficient quantities of bicarbonate of sodium and hydrochloric acid to cause effervescence.

Oertel's method combined systematic hill-climbing with a diet in which fluids and fat-forming elements were reduced to a minimum; but it cannot be recommended.

NEW GROWTHS AND PARASITES.

Under this head we may shortly mention *tubercle*, *cancer*, *syphilis*, *hydatids*, and *cysticerci*.

Tubercle.—Tubercles not infrequently form in connection with inflammation of the pericardium (*see* p. 543), when they are found as whitish-grey or yellowish granulations, mostly in the substance of the pericardial lymph or false membrane uniting the layers of the cavity, or sometimes actually under the layer of the visceral pericardium. They occur in the course of general tuberculosis, or at least secondarily to tubercle of the bronchial or mediastinal glands, or of the lungs and pleura. The diagnosis can only be made from the appearance of pericarditis under those circumstances; but it must be remembered that a pericarditis, which is not tubercular, may also arise in the course of phthisis. Isolated deposits of tubercle are exceedingly rare.

Cancer.—This attacks the heart in different forms, chiefly as lymphoma, sarcoma, and melanotic cancer. Frequently the heart is affected as a consequence of sarcoma or lymphoma of the mediastinal glands; the tumour then spreads along the veins, invades the auricles, and appears as nodular elevations under the pericardium. Sometimes the tumour is secondary to a similar growth in some other part of the body. A primary isolated deposit in the heart is exceedingly rare. There are no symptoms that are distinctive of cancer of the heart. It could only be inferred, in certain cases, from the existence of intra-thoracic growth. Walshe records a case where the conversion of the anterior wall of the right auricle into cancer was unattended with any clinical evidence pointing to the heart.

Syphilis.—The lesions of this disease occur as arteritis, as fibrous scars (*syphilitic myocarditis*), as fibroid masses, or as distinct gummata, which may be cheesy, and even softening in the centre, affecting the muscular substance of the heart in the same way as the voluntary muscles and causing some surrounding inflammation. The gummata are seated chiefly in the walls of the ventricles. They produce no characteristic symptoms, but may be the cause of irregularity, attacks of angina, or syncope, with evidence of enlargement if the myocarditis is extensive. Sudden death has rather frequently occurred.

Parasites.—*Hydatids* occasionally develop in the substance of the heart, forming cysts which project in the course of the growth either towards the pericardium, or into one of the cavities. The cysts are single, or may contain daughter-vesicles. Their effect [illegible] depends, of course, upon the size to which they [illegible] been found *post mortem* in death from causes independent of the heart; but in many cases they have been directly fatal, either by escaping from the wall of the heart into its cavity, or by rupturing and discharging daughter-cysts into the interior or into the pericardium. In the former case the daughter-cysts may get impacted in branches of the pulmonary artery, and cause death rapidly; or in some large artery of one of the limbs, and be followed by gangrene. A case is recorded of rupture at the same time into the ventricle and into the pericardial cavity, so that hæmo-pericardium resulted. Naturally the existence of a considerable cyst would interfere with the action of the heart or of its valves, but there is nothing by which such disturbance could be referred during life to hydatid, unless a cyst were known to exist elsewhere.

The *cysticercus* of the tænia solium is also sometimes found in the walls of the heart.

HYPERTROPHY AND DILATATION.

Hypertrophy is increase of size of the walls of the heart's cavities; dilatation is the unnatural distension of these cavities, and increase of their cubic capacity. The two conditions often co-exist, and hence one may have to deal with (1) simple hypertrophy, the cavity remaining of the normal size; (2) dilatation with thinning of the wall; (3) dilatation with hypertrophy.

The last occurs most frequently, hypertrophy predominating in some cases, and dilatation in others. Hence much of what is said under hypertrophy applies to cases of hypertrophy and dilatation when the former is in excess; and much of what is said under dilatation applies to the combined lesion where dilatation is the more pronounced.

HYPERTROPHY.

Hypertrophy arises in the muscles of the heart, as it does in the muscles of the body generally, from an increased amount of work, so long as nutrition is well maintained by a proper supply of good blood. All the cavities may be enlarged together, but it will be convenient to deal with them separately.

Hypertrophy of the Left Ventricle.—The left ventricle of the heart, as having the largest amount of work in propelling the blood through the extensive arterial system, is most often the subject of hypertrophy, and the increase of its work is most frequently due to some form of obstruction in the arterial system. The increased effort to overcome the obstruction results in hypertrophy. These causes of obstruction are:—(1) Disease of the sigmoid valves narrowing the orifice; (2) constriction of the arteries, either from atheroma or very rarely from congenital narrowness; (3) degeneration of the arteries (*arterio-sclerosis*) interfering with the free circulation of the blood. A very common cause of hypertrophy of the left ventricle is (4) Bright's disease, but the exact manner in which the hypertrophy is produced is still matter for discussion (*see* Cardio-vascular Changes in Bright's Disease). (5) Excessive action of the heart from exercise, from overstrain, and from palpitation, whether purely nervous, or as a part of the disease known as exophthalmic goitre, will also produce hypertrophy. Pregnancy, by the increased strain it throws upon the heart, has been credited with the production of hypertrophy, but it is at least doubtful. (6) Another cause is dilatation, which allows an abnormal quantity of blood to be present in the cavity, and hence increases the work which the walls of the cavities have to do in driving it out. (7) Mitral regurgitation is a cause of hypertrophy, by first leading to dilatation. Some doubt attaches to the view that (8) pericardial adhesions will cause it. They may be supposed to do so by opposing the contraction of the cavity, and thus increasing the work of the muscle; but they certainly in some cases are associated with dilatation from (9) myocarditis accompanying the pericarditis, and this dilatation it may be which results in hypertrophy.

Physical Signs.—The physical signs of hypertrophy of the left ventricle result from its enlargement and excessive action. In pronounced cases the impulse is forcible, and diffused over a wide area, so as to be manifest to the eye or the hand over a space of two or three square inches by a movement of elevation communicated to the ribs as well as to the intercostal space. It has often with this a slow or heaving character. The position of the impulse is altered, so as to be lower and farther out than the normal; the downward displacement being pronounced in hypertrophy from aortic disease; the outward displacement more marked under other conditions. The præcordial dulness tends to be increased in proportion to the enlargement. It may rise into the third intercostal space, may extend to the right over the right border of the sternum, and may reach externally one inch or more beyond the left nipple. It must, however, be allowed that in so much enlargement dilatation has a considerable share, and that in pure uncomplicated hypertrophy the increase of the præcordial dulness may be very slight. It must also not be forgotten that enlargement of the heart may be completely concealed by emphysema of the lungs, which may so cover it that both impulse and præcordial dulness are absent. The auscultatory signs are much less distinctive, as the conditions which so often cause the hypertrophy themselves modify the heart-sounds; for instance, disease of the valves, and altered conditions of the arterial circulation. Thus, valvular diseases give rise to murmurs, accompanying either the first or the second sound, as has already been explained; and in Bright's disease the cardiac sounds are habitually modified by the alterations in the arterial tension which result from it. It is commonly said that in simple hypertrophy the first sound is less loud than normal, or muffled, and this is attributed to the thickness of muscle through which the sound from the closing auriculo-ventricular valves has to travel. The pulse, like the heart-sounds, is mostly affected by the causes of the hypertrophy rather than the hypertrophy itself, and presents the characteristics of aortic obstruction, or regurgitation, or mitral disease, or Bright's disease, or atheroma, as the case may be. In proportion to its dependence upon the heart it will be full, strong, tense, and incompressible.

Symptoms.—These are a sense of discomfort, or actual pain about the præcordia, from the forcible beating of the heart, increased by exertion; dyspnœa on exertion; and sometimes syncope. The patient's face is said to be flushed; and headache, noises in the ears, flashes of light, mental dulness occur as though from increased tension in the cerebral circulation. Probably hypertrophy of the heart leads to degeneration of vessels, and hence in some cases to their rupture. Where the hypertrophy results from disease of the valves it will only be sufficient to overcome the obstruction at the orifice, and hence the arteries are not subject to undue pressure. It is different if hypertrophy arises from capillary obstruction; the arteries become in due time degenerated, and they may give way. But it is doubtful if a hypertrophied ventricle could rupture healthy arteries.

Diagnosis.—The conditions with which hypertrophy of the left ventricle is likely to be mistaken are the following:—1. Over-action of the heart from *excitement.* This condition is often seen in young people apparently well, examined perhaps for an appointment or for life insurance. The action of the heart is here generally rapid, the beat is quick and sudden, rather than slow and heaving, the patient is obviously nervous, and the condition is easily shown to be merely temporary. A murmur is sometimes heard under such circumstances (*see* p. 525). 2. The heart may be uncovered from *retraction of one lung*, usually the left; and a greater surface being in contact with the chest, it may give increased præcordial dulness, and more extended impulse. The normal position of the apex beat, with the absence of forcible heaving, will distinguish this condition. 3. *Pericardial effusion* is frequently confounded with hypertrophy and dilatation, perhaps more often with the latter (*see* Pericarditis). 4. Displacement of the heart from new growths, or pleuritic effusion. 5. Aneurysm. 6. In all enlargements of the heart the relative share of hypertrophy and dilatation must, if possible, be estimated. Probably no considerable enlargement takes place without dilatation. Moderate enlargements may arise acutely from dilatation. Dilatation is greater, the more feeble the impulse.

Prognosis.—Hypertrophy, due to causes which are not permanent, such as excessive exertion, has improved in the course of time. But with a persistent cause like valvular disease or arteriosclerosis, no permanent recovery can be looked for, and it is much more probable that dilatation will sooner or later become a prominent feature in the case.

Treatment.—The removal of the cause must be considered. If this consists in continued overstrain, or excessive and unsuitable food, by which the arteries tend to degenerate, much may be done by rest of mind and body, by a properly regulated diet, by being careful not to overload the stomach, and by attention to the bowels; and in all cases the same principles should be followed out as a help towards improvement. With regard to drugs, it is not to be supposed that any will directly promote the diminution of the hypertrophy; but the action of the heart may be quieted when necessary by the cautious administration of the cardiac depressants, aconite and veratrum viride: from 2 to 5 minims of the tincture in each case may be given. Digitalis is of more value where dilatation accompanies the hypertrophy, and is sometimes actually harmful where hypertrophy is largely predominant.

Hypertrophy of the Right Ventricle.—This arises, in the majority of cases, from obstruction to the pulmonary circulation, (1) at the pulmonary orifice, from congenital malformations of the valves, congenital constriction of the orifice, acquired disease of the pulmonary valves, or pressure on the base of the pulmonary artery by aortic aneurysm; (2) in the lungs, from emphysema, chronic bronchitis, bronchiectasis, or occasionally chronic phthisis; and (3) from primary disease on the left side of the heart, whereby the left auricle becomes engorged, and the pulmonary venous circulation is consequently impeded. The signs of hypertrophy of the right ventricle are analogous to those of left-side hypertrophy. A marked impulse may be seen in the epigastrium, due often to the impact of the right ventricle against the liver, rather than to direct contact of the ventricle with the abdominal walls at this point; the apex-beat may also be carried somewhat to the left. The præcordial dulness extends beyond the sternum. The pulse, if affected at all, is small on account of the difficulty the blood has in reaching the left side of the heart; or it is modified by the condition of the left ventricle. The sounds are not materially altered. Dyspnœa is present, often from the condition of the lungs.

Hypertrophy of the Auricles.—This rarely occurs without dilatation but predominates in the left auricle in mitral stenosis

(Samways). It arises from constriction or incompetence of the auriculo-ventricular valves, or from hypertrophy and dilatation of the ventricles acting back upon the auricles. Some of the forcible impulse in mitral stenosis is attributed by Sansom to the action of the hypertrophied left auricle, and variations of the mitral obstructive murmur are partly due to the same.

DILATATION.

Ætiology.—The causes of dilatation of a given cavity of the heart are, on the one hand, those which tend to produce over-distension of the cavity; on the other, those which diminish the strength of the walls of the cavity, so that they yield to the force which is distending them. The cavity becomes over-filled if the blood is too quickly poured into it, as in aortic regurgitation, or if there is any obstacle to its free exit through the natural channels. In this last way nearly all the causes of hypertrophy enumerated for the different cavities of the heart are causes also of dilatation. Of these, valvular diseases, and high arterial tension in Bright's disease, are the most common. The yielding of the ventricular walls is promoted by the various degenerative changes, some of which have been described; the fatty degenerations, fibroid degeneration, degenerative changes of old age, the granular or fatty conditions which follow upon anæmia, chlorosis, and severe and prolonged fevers, and the myocarditis and pericarditis of acute rheumatism. The excessive indulgence in alcohol is sometimes a cause of cardiac dilatation with all its consequences. Many of these conditions are opposed to the full development of hypertrophy in the event of an obstruction to free circulation. But such predisposing causes are not essential. With considerable obstruction, the strength of the healthy muscle may be overpowered before hypertrophy has time to take place, at least in a degree sufficient to prevent dilatation.

Anatomical Changes.—The effects on the size and shape of the heart vary with the cavity concerned. In general dilatation the heart becomes more globular, and is widened transversely. The dilated left ventricle increases to the left; and the dilated right ventricle often bulges out so as to make a prominent angle on the normally curved right border of the heart, and pushes itself in front of the left ventricle at the apex. The thickness of the walls will depend on the presence or absence of accompanying hypertrophy. In dilatation with thinning, the ventricle walls may be reduced to one-sixth of an inch, and even less at the apex, which is commonly the thinnest part. The auriculo-ventricular orifices share in the dilatation, and incompetence often results.

The consequences of dilatation are of much importance. Whereas hypertrophy is compensatory and preservative, is developed in proportion to the work the heart is called upon to do, and, in the majority of cases, does not react injuriously upon the circulation, dilatation is only the sign of the weakness of the propelling organ, and has for its effects the retardation of the flow of blood throughout the system. The dilatation of the left ventricle, with imperfect emptying of its cavity, is followed by incompetence of the mitral valve, by dilatation of the left auricle, and this in succession by engorgement of the lungs, increased tension in the pulmonary arterial system, dilatation of the right ventricle, incompetence of the right auriculo-ventricular (tricuspid) valve, dilatation of the right auricle, and congestion of the systemic venous circulation, with its secondary results—dropsy, venous congestion of the liver, spleen, and kidneys, albuminuria, and other effects, which are described in more detail under the head of "Diseases of the Valves."

Physical Signs and Symptoms.—To a much greater extent than in simple hypertrophy, the enlargement of the heart has its influence on the extent of præcordial dulness, and on the position of the impulse. The dulness is increased to the left or the right according to the cavity mainly involved; a larger portion of the heart comes into contact with the chest-walls, so that the ventricular movements are seen in two or more intercostal spaces. The impulse is carried outwards, and a little downwards. The apex-beat is generally feeble, and in some cases scarcely visible or palpable; and it is irregular, either constantly or only when additional stress is put upon the heart. The sounds in extreme cases are very faint, though clear; in others the first sound tends to be short, clear, and somewhat high-pitched; and the second is but little affected. But just as in hypertrophy, the sounds will often depend on the valvular or arterial conditions which have helped to produce the dilatation.

Dilatation of the Left Ventricle is often accompanied by a systolic murmur, which is mostly high-pitched, of short duration, and localised to the neighbourhood of the apex. Opinions are rather in favour of this murmur being produced by vibrations in the cavity of the ventricle than of its being a murmur of mitral regurgitation. Mitral regurgitation is, however, one of the effects of dilatation, and may be accompanied by its characteristic murmur, audible in the back. The pulse is affected by the inefficiency of the ventricle—it is small, feeble, and perhaps irregular. In extreme cases the impulse is much displaced and may be felt even in the axilla, three or four inches to the left of the nipple. The general effects of dilatation are—shortness of breath, palpitation, and more or less cardiac distress on exertion; occasional attacks of syncope; a moderate degree of anæmia in many cases, with a not quite perfect nutrition. Digestion is also often impaired.

Dilatation of the Right Ventricle causes increase of the præcordial dulness to the right of the normal position; but also largely to the left; at the same time there may be visible pulsation in the second, third, and fourth left intercostal spaces, and in the epigastrium, often with an undulating movement, the lower spaces retracting while the upper rise. In advanced conditions it leads to a tricuspid regurgitant murmur (pp. 517, 523), and is accompanied by the indications of congestion of the systemic venous circulation, which have been described (p. 511). A dilated right ventricle possibly contributes to the causation of pulmonary murmurs in anæmia and other conditions (*see* Anæmia).

Dilatation of the Left Auricle causes sometimes dysphagia from pressure on the œsophagus, and may compress the left bronchus in extreme cases. Enlargement of the *right auricle* causes increase of the præcordial dulness towards the right of the sternum; and, it is said, may give rise to an impulse in that situation, preceding the systole.

Diagnosis.—Dilatation has an important share in all the considerable enlargements of the heart, and it may be confounded with the several lesions mentioned under hypertrophy (p. 543). The chief physical evidence is the displacement of the impulse towards or beyond the nipple-line; and if that is produced by pleural effusion on the right side, or by aneurysm, some other proof of the existence of such a lesion will probably be afforded. The triangular dulness of pericardial effusion may be like that of a dilated right ventricle, but the impulse is raised by liquid into the fourth or third space beneath or above the nipple, and not pushed below and outside it. Further distinctions may be found in the associated vascular conditions, and in the history or presence of a rub.

Prognosis.—This is unfavourable. Recent dilatation from acute diseases or other temporary cause may recover; but in dilatation of any standing the treatment can only be palliative. The best thing that can happen is the development of hypertrophy.

Treatment.—This is, in the main, the same as that of valvular diseases—rest; light but nourishing diet; the use of digitalis or strophanthus to give tone and strength to the heart-walls, combined in severe cases with direct stimulants, ammonia and brandy; and diuretics and purgatives to relieve systemic venous engorgements. In conditions of great distension of the right ventricle, venesection may be required to prevent the cessation of the heart's action. In earlier stages the general conditions, such as anæmia, and the tendency to formation of fat, which have favoured dilatation, may be dealt with by suitable means.

The methods of Schott, above mentioned (p. 539), may be useful in some of these less severe cases.

PERICARDITIS.

Ætiology.—Inflammation of the pericardium may result from a general blood-poisoning; or it may occur from direct irritation or infection of the serous sac.

Among the first class of cases, acute rheumatism is its most frequent cause; it occurs in Bright's disease, in pyæmia, in leuchæmia, in tuberculosis, in influenza, and in other conditions of toxæmia. Its local causes are the growth of cancer nodules into its cavity, the rupture into it of abscesses and hydatid cysts, and the contiguity of inflammation of the respiratory organs, whether empyema or pneumonia.

Anatomical Changes.—If we take as the type the pericarditis which occurs in the course of acute rheumatism, we find the following changes:—In early stages the membrane loses its smooth, glossy surface, and becomes more vascular, so that it is injected with a fine network of vessels. Some shreds of lymph from the exudation of corpuscular elements and fibrin from the blood-vessels are next seen, and a complete layer forms upon the pericardium. Ultimately the two opposed surfaces of the sac may be separated by a layer of lymph one-eighth or a quarter of an inch in thickness, which is sufficiently soft to allow the parietal and visceral membranes to be peeled from one another, and the lymph is often of such a consistence that the separation of the surfaces leaves a curiously honeycombed or reticulate appearance. Generally, at the same time, some serum is formed, of yellow colour, and turbid from corpuscular elements. This may accumulate to a considerable amount and further separate the two layers of the pericardium, while it allows the formation of long shaggy processes of lymph, stretching from surface to surface. After a time the fluid generally disappears, and the lymph is either itself absorbed, or it becomes organised, and unites the parietal and visceral layers of the sac more or less completely together. In this process new vessels grow in the investing lymph, and fibres of connective tissue are gradually developed. The amount of firm connective tissue thus formed and the completeness of the union effected vary much in different cases; there may be a few fibrous bands crossing the cavity, or a dense layer of tissue half an inch thick.

Variations in this process take place. Under certain circumstances, mostly in pyæmia or septicæmia, the fluid contents of the pericardium are pus, instead of serum, constituting *purulent* or *suppurative pericarditis*. This is often secondary to abscess of the cardiac muscle, which is known frequently to result from acute necrosis of the long bones. Sometimes the new-formed vessels in the inflammatory formation rupture, and small petechiæ or larger patches of hæmorrhage cover the surface of the membrane, forming *hæmorrhagic pericarditis*. And, occasionally, tubercles are formed both in the new tissue and in the original membrane covering the heart's surface; this is known as *tubercular pericarditis*, and forms part of a general tuberculosis.

There are no micro-organisms peculiar to pericarditis; streptococci and staphylococci are most common, and pneumococci or tubercle-bacilli occur in special cases.

Symptoms.—Since pericarditis so frequently arises in the course of some general disease, like rheumatism, its symptoms may be entirely masked by those of the disease which it accompanies, and its presence may be only revealed by the alteration in the heart-sounds, and other physical signs which it produces. These, however, are generally characteristic. In the early stages a slight shuffling sound is heard in addition to, though partly obscuring, the normal sounds. The shuffling consists of two sounds occurring during systole and diastole respectively, but not always absolutely synchronous with the first and second sounds; it is heard over the præcordial region, often first at the base, later over the whole of the area corresponding to the anterior surface of the heart and pericardium. Frequently this *pericardial rub* has a triple character, like the pace of a cantering horse. After a short time the sound becomes louder and harsher, resembling the friction of hard rough surfaces together, and when it has reached this stage the friction can often be felt by the hand placed over the præcordial region.

If liquid is effused into the pericardium, as is frequently the case, the præcordial dulness is increased. It extends upwards to the upper border of the third rib, the upper border of the second rib, or even to the clavicle; towards the right for one inch or more over the sternum; and towards the left for an inch or so beyond the left nipple. The præcordial dulness has a more or less triangular shape, with its broad base upon the diaphragm, and a rounded apex at the upper part of the sternum and the left upper intercostal spaces. As the fluid increases, the impulse of the heart moves gradually outwards and upwards, until with much effusion it may be felt in the third left space above and external to the nipple; this is not because the apex is raised, but because the impulse is formed by a part of the heart more and more remote from the apex. An important distinction must here be mentioned between pericarditis and pleurisy in the effect of effusion of fluid on the occurrence of a friction murmur. In pleurisy the effusion of fluid results in the disappearance of the pleuritic friction sound. In pericarditis, the friction sound persists commonly throughout the illness, even to the period of greatest distension of the sac, and during the subsequent absorption of the fluid. In the case of the pleura the effusion compresses the lung and separates the two pleural surfaces from one another; in the case of the pericardium, the effusion finds less resistance on the side of the lungs, and thus increases præcordial dulness, while between the solid heart and the spine and sternum, behind and in front, there is but little room for the accumulation of a large quantity of liquid.

The local symptoms which may accompany these conditions are pain, anxiety or distress at the præcordia, tenderness on pressure over that region, shortness of breath, with shallow respirations, and short, hacking cough. The pulse may not at first be much affected, but it tends soon to be faster and fuller, and in the later stages of effusion to become feebler, and even fluttering and irregular. Occurring in the course of a febrile disease like rheumatism, it may not notably add to the existing pyrexia, but its rapid onset occasionally causes considerable elevations of temperature—for instance, to 105° or 106°; and in other cases it tends to produce the usual conditions of pyrexia, loss of appetite, dry tongue, thirst, and scanty urine.

In the worst cases the cardiac feebleness increases, the pulse becomes irregular and fluttering, præcordial anxiety is severe, and the face becomes drawn and pinched; and the dyspnœa is perhaps further aggravated by the pressure of the distended pericardial sac upon the left lung, which may be so compressed as to lead to dulness and bronchial breathing at the left base below the angle of the scapula (*see* p. 436). Ultimately, the nervous system fails, and delirium, jactitation, convulsion, or coma ends the scene. But in the majority of instances the symptoms gradually subside; the dulness diminishes from above downwards, while the rub often remains till a late stage. In many cases, no doubt, adhesion of more or less of the pericardial surfaces takes place.

The changes of pericarditis occur rapidly, effusion may reach its height in two or three days, and subsidence may be well established in three or four more.

Suppurative, tubercular, and hæmorrhagic pericarditis are not essentially different in their symptoms and physical signs.

Diagnosis.—Under ordinary circumstances this presents no difficulties, the double or triple friction sound being very distinctive. A double pericardial rub may, however, sometimes be simulated by a double aortic murmur, and if there is simultaneous dilatation of the right ventricle, the outline of pericardial effusion may be closely simulated. The co-existence of rheumatic fever will not always help, as it might accompany both pericarditis and old aortic disease. The pericardial friction sounds are, however, less likely to be limited to the area of aortic valve murmurs; they are often not strictly synchronous with the heart-sounds, are not accentuated at the commencement, are often increased by pressure with the stethoscope, and vary within a few hours in their relative intensity at different parts of the præcordia.

Prognosis.—Pericarditis is not, on the whole, an immediately fatal disease. It may be so mild that it is only detected by the stethoscope in the course of a routine examination, and in a large proportion of the cases occurring in rheumatic fever the inflammation subsides. The adhesion of the layers, which so often results, may become a danger in itself, but it must not be forgotten that pericarditis in acute rheumatism is often accompanied by, and masks, some inflammation both of the endocardium and of the muscular substance of the heart, the ill effects of which become developed afterwards. In Bright's disease, and in association with other chronic cachexiæ, it often occurs towards the end of the illness, and then may appear to be the lesion determining death; but even under such circumstances the physical signs may completely disappear before death, or, if they persist, the fatal result may not seem to be hastened thereby. The recognition of the associations suggestive of tubercular or purulent pericarditis will make the prognosis a grave one.

Treatment.—The treatment of pericarditis is mainly palliative. Like other acute inflammations, it must be met by complete rest in the recumbent or semi-recumbent posture, by nutritious fluid diet, and by abstinence from talking, excitement, or worry. In the case of rheumatic fever, these conditions are probably already provided in the treatment of the initial disease, which may be continued, provided that the heart's action is not seriously weakened by any drug—*e.g.*, salicylates in excess. The further indications required by the implication of the heart resolve themselves into the relief of pain, the maintenance of the strength of the heart and circulation, and the absorption of effused fluid where this subsides slowly. In very severe pain, six or eight leeches may be applied to the præcordia; but the same effect may be obtained by the use of morphia internally, or by subcutaneous injection. The præcordia may be protected by a layer of cotton wool, or a warm linseed-meal poultice may be applied. If the circulation tends to fail, or the heart becomes irregular, small doses of tincture of digitalis should be given frequently, with brandy or ammonia. Medicinal measures for the removal of fluid in pericarditis are uncertain. Mostly it begins to be absorbed if the heart can be sustained during the short period when it is at its height. If a considerable quantity remains for some time, iodide of potassium may be given. If the liquid is in excessive amount and threatens a fatal result, or if it is presumed to be purulent, paracentesis may be performed. The fifth left intercostal space is usually selected; an incision is made through the skin one inch from the sternum, and a trocar and cannula are then inserted. The fluid may be aspirated if desired.

ADHERENT PERICARDIUM.

Reference has been already made to this condition as arising from pericarditis. The degree to which the two surfaces may adhere varies much in different cases; there may be merely a few filaments running from the surface of the heart to the parietal pericardium, or there may be complete union of the pericardial sac to the surface of the organ, and every intermediate condition occurs. When the union is complete, the tissue uniting the two surfaces may form only a thin layer; or it is a dense, firm, fibrous, more or less vascular coat, a quarter or even half an inch in thickness. In rare cases, also, calcareous matter is deposited in the adhesions, so that a complete investment by it may take place. Although the serous sac of the pericardium seems specially devised to allow the free movement of the heart, the simple adhesion of the two layers is not necessarily followed by any ill effects upon the form and size of the heart, and in a certain proportion of cases the heart has its normal size. But in others hypertrophy or dilatation is present. In many of these last valvular disease is associated with the pericarditis, and sufficiently accounts for the changes in the heart-walls. If the pericardial adhesion is very extensive and dense, dilatation and hypertrophy may occur without being accounted for by any valvular disease, and it is here probable that the muscular substance of the ventricle has been injured by the occurrence of myocarditis at the same time as the pericarditis.

Symptoms and Physical Signs.—The symptoms associated with pericardial adhesions are for the most part, if not entirely, due to dilatation and hypertrophy of the cardiac walls: anginal pain, palpitation, and dyspnœa may be especially mentioned. Their recognition depends more often upon the history of previous inflammation, and the existence of certain physical signs, which are by no means always present, and which require not only adhesion of the pericardial surfaces, but also fixation of the pericardial sac to the wall of the chest by means of an adherent pleura. The physical signs upon which most stress has been laid are—(1) systolic retraction at the point corresponding to the apex of the heart; (2) systolic retraction of the lower end of the sternum; (3) systolic retraction of the third, fourth, or fifth intercostal spaces to the left of the sternum; (4) systolic retraction of the lower ribs at the side or back of the left chest (J. Broadbent); (5) absence of alteration in the præcordial dulness, and in the position and force of the impulse during respiratory movements; (6) sudden collapse of the veins of the neck during ventricular diastole (Friedreich). Certainly systolic retraction of intercostal spaces is not peculiar to adherent pericardium, much less is systolic recession of the epigastrium; some other signs are difficult to verify in particular cases, others are not constantly present. Since adherent pericardium so often occurs in company with valvular and myocardial lesions, the physical signs, as well as the symptoms, of the latter are apt to be credited to the former. Adhesions may sometimes be inferred, when the patient is suffering from evidence of failure of the right ventricle, such as dyspnœa, dropsy, enlargement of the liver, and albuminuria, without any obvious cause for the right-sided failure, such as mitral or pulmonary disease. This diagnosis is still more reasonable if there is or has been pleurisy or pleuritic effusion or adhesions on one or both sides, as this increases the probability of a combined pleural, pericardial, and mediastinal inflammation having been present.

The **Prognosis** and **Treatment** of pericardial adhesions can only be considered in reference to the changes in the structure and functions of the heart which result from them (*see* Hypertrophy and Dilatation).

PERICARDIAL EFFUSIONS.

HYDROPERICARDIUM.

This term is intended to denote the presence of an excess of serum in the pericardial sac, and is generally used to distinguish the passive secretion of dropsy from that of inflammatory effusions already described under Pericarditis. The pericardium naturally contains a very small quantity of serum, and after death from any cause it is common to find a few drachms of pale yellow fluid in it. When this reaches five or six ounces or more it constitutes dropsy of the pericardium, or hydropericardium. The causes of serous effusion, apart from inflammation, are those of general dropsy, such as Bright's disease, and such local interference with the venous circulation of the pericardium as valvular disease of the heart itself, chronic lung disease, and pressure of growths upon the veins which return blood from the pericardial surfaces. The liquid contained in the sac resembles that of dropsical effusion into the other serous cavities, being pale yellow, or more or less pink from exudation of blood-colouring matter, with a small quantity of fibrinogen, and from one to three per cent. of albumen.

The **Physical Signs** of hydropericardium are the same as those of effusion in pericarditis. As a rule, no special **Treatment** directed to the pericardium is required where the condition forms part of a general dropsy, or where it results from local interference with the circulation; the general dropsy or the valvular disease must be dealt with. In some rare cases the effusion may be so rapid or abundant as to require paracentesis of the pericardium.

PNEUMOPERICARDIUM AND PNEUMO-HYDROPERICARDIUM.

These signify respectively the presence of gas, and the presence of gas and liquid together, in the pericardium. Gas in conjunction with liquid has been observed as a result (1) of decomposition of the liquid of pericarditis; (2) of the communication of the pericardial sac with air-containing cavities. This communication may be traumatic, as in the case of a juggler, who, in attempting to swallow a blunt sword, perforated the pericardium from the œsophagus; as in the case recorded by Flint, where the pericardium was punctured by a stab through the pleura; and after the operation of paracentesis pericardii. Or the communication may be effected by disease; and cases are on record of cancer of the œsophagus ulcerating into it, of a phthisical cavity opening into it, and of a hepatic abscess communicating at the same time with the pericardium and with the stomach. Gas can never be observed alone in the pericardium, as its entrance from without is always immediately followed by pericarditis with liquid effusion.

The **Physical Signs** of pneumo-hydropericardium are resonance on percussion over the præcordial area, and splashing, churning, or gurgling sounds, synchronous with the movements of the heart. This last sign may be absent where the gas is largely in excess of the liquid; but in a case recorded by Walshe there was this peculiarity, that on placing the patient on one side the resonance was accompanied by an area of dulness due to liquid at the lowest part, and on turning him to the other side the resonance and dulness changed their relative positions. A few cases of recovery are recorded.

HÆMOPERICARDIUM.

In slighter degrees, the effusion of blood into the pericardium occurs in so-called hæmorrhagic pericarditis, from the rupture of the new-formed vessels; but larger quantities, when not directly traumatic, result from rupture of the muscular substance of the heart, of an aneurysmal sac, or of vessels in a cancerous growth. Scurvy and allied conditions may also give rise to pericardial hæmorrhage.

Symptoms.—When sudden effusion of blood into the pericardium takes place, the patient is seized with more or less oppression of the chest, pallor, syncope, unconsciousness and death in quick succession; or with the same pallor and with collapse, feeble pulse, dyspnœa, and orthopnœa, he may remain for twenty-four or thirty-six hours before the fatal termination; or presumably, with a less degree of hæmorrhage, death may be still further delayed and a pericarditis may develop, which contributes to the final result. Walshe refers to cases, probably of a scorbutic nature, or at any rate not dependent on rupture of aneurysms, or of the heart itself, in which recovery has taken place.

The **Physical Signs** are those of a large pericardial effusion; extensive præcordial dulness, and enfeeblement or absence of the heart-sounds. The **Diagnosis** would be assisted by a knowledge of the previous existence of aneurysm. Absolute rest and judicious use of stimulants would give the only chance in **Treatment.**

ANGINA PECTORIS.

This name is given to an intense pain in the region of the heart, which comes on with great suddenness, and occasionally proves fatal. In its most typical form it is a rare complaint.

Ætiology.—It scarcely occurs below middle age, and it is very much more frequent in men than in women, in the proportion of ten to one. As a large majority of the cases present some lesion of the heart or arteries, the conditions which lead up to these changes may be regarded as predisposing causes of angina pectoris; and especially obesity, sedentary occupations, and the gouty habit. Heredity also seems to have influence. The immediately exciting causes are mostly such as may be supposed to act prejudicially upon the cardiac functions, whether through its muscular or nervous apparatus. The most frequent are physical exertion, especially going uphill or against the wind, and emotional excitement, whether depressing or exhilarating. Much slighter exertion, or exposure to cold is sometimes sufficient; and occasionally the attack begins during sleep.

Symptoms.—The patient is seized quite suddenly with acute pain at the lower end of the sternum, rather to the left side; the pain radiates thence to the left side and back, or through to the scapula; up to the left shoulder, and down the left arm to the hand; or less frequently to the right shoulder, arm, and hand. Tingling or numbness may accompany the pain in the fingers, with this there is a feeling of tightness of the chest, or suffocation, and even of impending death; but there is no dyspnœa of the usual type. The patient is obliged to stop if he is walking; he becomes collapsed, faint, and covered with clammy perspiration. The behaviour of the heart seems to be variable; at any rate accounts differ widely. The pulse is sometimes, but not always, irregular—it may be scarcely altered in frequency, and is not necessarily weak. Sometimes the pulse is increased in tension at the time of the attack; but this is not a universal rule. The attack is often accompanied by flatulence, and followed by the passage of abundant pale urine. After lasting a few seconds or minutes, the pain quickly passes off, but it may recur again frequently in the course of a few hours, or it may not be experienced again for several months or years. Angina may be fatal in the first and only attack.

Slight attacks of cardiac pain occur in some patients, which have not the dramatic features of the case first described; they do not, however, differ materially from true angina. They depend on similar arterial or cardiac lesions, and are cured or relieved by the same means.

Pathology.—When death has taken place in an attack, the heart has generally been found relaxed, wich its cavities full of blood. In the majority of cases, some disease of the heart or aorta has been found, and mostly of the following kinds: fatty degeneration of the myocardium; atheroma or calcareous change in the aorta or its valves; aneurysmal dilatation of the aorta; and lastly, atheroma or calcareous deposit in the coronary arteries, or their obliteration from these or similar causes. Clinically, also, it appears that in a large proportion of cases some auscultatory evidence of one of the above cardiac defects is forthcoming. Disease of the mitral orifice, on the other hand, is rarely the sole cause of angina pectoris.

The predominance of these lesions is one strong argument against the disease being purely neuralgic: but it is not at present certain what is the nature of the pain, and how it is brought about. The most probable view is that it is due to a degenerated or feeble heart striving to overcome a high, or suddenly increased, peripheral resistance in the small arteries. A consideration of the tension of the pulse led Brunton to administer nitrite of amyl, with the object of diminishing it; and the angina was promptly cured. The successful treatment of angina by nitrite of amyl has been experienced numbers of times since, but the arterial tension is not always high. Liégeois and others refer angina to an *ischæmia*, or anæmia of the heart's substance, consequent upon the actual constriction of the coronary arteries in some cases, and upon imperfect blood-supply through them in others—*e.g.*, free aortic regurgitation; and he explains the effect of nitrite of amyl by its dilating the vessels, and thus flushing the myocardium with blood. Cramp of the cardiac muscle has been suggested, but the continuance of the pulse seems opposed to this.

Diagnosis.—The character of the pain, the cause of its occurrence, and the evidence of a cardiac or arterial lesion, are generally conclusive. It has to be distinguished from neuralgic pains, especially in neurotic women, in whom actual lesions are not present, though the pain may be accompanied by signs of vasomotor constriction (coldness and numbness of the extremities). Osler attaches much importance to the valvular lesions and arteriosclerosis.

Prognosis.—In its severer forms this must be grave, as there is always a fear of recurrence, which may be too quickly fatal for treatment to be of any avail. But milder cases are often amenable to treatment.

Treatment.—Undoubtedly the most efficacious remedy for an attack of angina is nitrite of amyl, which is conveniently carried in small glass capsules, each containing from three to five minims; one of these is crushed between the folds of a handkerchief, and the vapour is inhaled freely. The effect is to dilate the peripheral arterioles, the face flushes, the cranial vessels throb, and the pain is often relieved at once. The dose may have to be repeated. A similar effect may be obtained by the administration of nitro-glycerine internally, but its action is not so rapid. One may begin with $\frac{1}{100}$ minim, given in the form of tabella; or with a one per cent. solution in alcohol, of which one minim in a little water is the required dose. Much larger quantities may have to be given, equivalent to two, three, five, or ten-hundredths of a minim. The first administration of even small doses of nitro-glycerine is often followed by a throbbing headache, but after a time tolerance is established and the larger doses can be borne. Sodium nitrite (2½ gr. in tabella) and erythrol tetranitrate (1 gr. in 1 drachm absolute alcohol suitably diluted) are also good vaso-dilators. If these measures fail, the hypodermic injection of morphia, or the inhalation of chloroform, may be used; and much collapse will require brandy or ether. When angina has once declared itself in a patient, nitro-glycerine should be given for several weeks; the dose may be $\frac{1}{100}$ minim three or four times daily, gradually increased to $\frac{1}{20}$ or $\frac{1}{10}$. Iodide of potassium (5 to 30 grains) is also beneficial in some cases. Arsenic and iron are valuable as cardiac tonics. At all times undue exercise, sudden movements, excess in eating and drinking, and mental excitement should be avoided.

FUNCTIONAL DISORDERS OF THE HEART.

The functional disorders of the heart, which we may here shortly consider, are frequent action, slow action, intermission of the heart's beat, and palpitation. All these may be seen in various forms of cardiac disease, but are sometimes present temporarily, or in paroxysms, when no organic disease of the heart can be discovered.

FREQUENT ACTION OF THE HEART.

This is natural to some persons; it occurs on exertion, and especially during excitement, in very many people. It is always important to remember this, both when consulted for actual illness and when asked to examine a candidate for life insurance; the pulse in such persons may run up at once to 120 or 140, though they are at the time in perfect health. Occasionally one meets with cases of paroxysmal increase in the rapidity of the pulse, lasting a few minutes to several hours (*paroxysmal tachycardia*). The attack may come on without any warning, and may cease as suddenly as it began; and it is sometimes accompanied with but little discomfort to the patient. Such attacks appear to be due to mental excitement, excessive physical exertion, or gastric disorder; it is occasionally impossible to trace them to anything of the kind. They occur both in males and in females, and probably a neurotic or hysterical condition predisposes to them. All the cardiac nerves have in turn been charged with the causation of this disorder, but nothing is really known about it.

Treatment.—Most cases are benefited by digitalis, which should be given during the attack; but one should try to prevent the return of the paroxysm by rest, by the avoidance of all excitement, by careful attention to diet, by abstinence from tea, coffee, and tobacco, and by a course of potassium or ammonium bromide in moderate doses. Electrical stimulation of the vagus in the neck has been found useful. In some cases the tachycardia yields to morphine injections.

INFREQUENT ACTION OF THE HEART.

This condition (*bradycardia*) is also peculiar to some people throughout life. It accompanies sclerosis of the coronary arteries and some other lesions which have already been mentioned (p. 494) and occurs as a result of, or in the course of, numerous disorders, such as pain, exhaustion, convalescence from acute disease, hysteria, mental disorders, toxic states, &c. The actual heart-beats, as well as the radial pulse, should always be counted.

INTERMISSION OF THE HEART'S ACTION.

The intermittent pulse, already described (p. 495), is due to a corresponding cessation of the heart's action. There is at the same time a sudden and distressing sinking sensation in the cardiac region, or a feeling as if the heart fluttered or tumbled over.

Ætiology.—Intermission occurs at almost any adult age. In younger persons it is often traceable to gastric disturbance, or to the too free indulgence in tea, coffee, or tobacco. Overloading the stomach is a common cause, and it frequently occurs in the early morning hours after a heavy supper or late dinner. In such a case it ceases as the stomach disposes of its contents. When the intermission is due to tea or tobacco, or repeated indigestion or flatulence, it is more persistent, and it may be present day after day until the cause has been discovered and removed. In persons past middle life, intermission is apt to be troublesome for long periods, or even to the end of life. In some cases the above-mentioned causes may be at work; in others there may be evidence of senile changes such as atheromatous arteries; in others, again, no appreciable alteration in the heart, arteries, or other organs can be detected.

The **Prognosis** is much more doubtful in these cases than in those of younger patients. Many people, it is true, live on without further indications of cardiac change; but in those with arterial degeneration the symptom must not be ignored.

Treatment.—In young people, tea, coffee, and tobacco should be forbidden, if either of these can be shown to cause the trouble. All mental worry or physical overstrain should be avoided; the diet should be carefully attended to; the digestion should be assisted, and flatulence prevented, by bismuth, sodium bicarbonate, spiritus ammoniæ aromaticus, and calumba or gentian. Diffusible stimulants, such as ether, ammonia, and alcohol, are likely to diminish intermission for a time; but ammonia is the only one that can be safely used continuously.

PALPITATION.

By this term is meant an unduly forcible action of the heart perceptible to the patient.

Ætiology.—As a functional disorder, it is especially common in nervous hysterical females, but occurs in both sexes as the result of depressing influences upon the nervous system, such as mental worry and excitement, over-study, exhausting illnesses, anæmia, and excessive smoking or tea-drinking. It is paroxysmal in its occurrence, and the attacks may be induced by excitement, physical exercise, overloading of the stomach, or indigestion.

Symptoms.—During a severe attack there is much distress from the violent beating of the heart against the chest-wall, the carotids throb, there is a sense of faintness, or a fear of impending death. The heart's impulse is in the natural position, but the beat is widely diffused; the præcordial dulness is not increased, and the pulse is not necessarily quicker than normal. The cardiac sounds are loud, clear, and ringing, and there is no murmur

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two or three hours, combine
bromide; or with valerian, asa
in the usual doses.

DISEASES OF THE

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embolism will also be considered in t

ARTERIT

Ætiology.—The following are re
inflammation:—(1) Local contact wi
foci, as in arteritis from suppuration
which acts especially as a cause of inf
large vessels; (3) syphilis, affecting
and also vessels actually in contact
(4) tubercle, which attacks the vess
invades. In tubercular meningitis
attacked; and in phthisis the tube
wall of the vessels, leads to
aneurysm.

acute aortitis also occurs, in which all the coats of the vessel become thickened with cellular infiltrations, especially about the vasa vasorum, as well as on the surface, and in the meshes of the inner coat. In *chronic arteritis*, which may begin as an acute disease, the inner surface presents broad greyish-white slightly raised patches, which may be soft, mucoid, and gelatinous, or more or less sclerosed from the presence of fibrous tissue. In later stages there may be a semi-cartilaginous thickening, affecting the inner coat mainly. But the same changes involve also the middle coat, destroying the muscular and elastic elements, and the outer coat, causing fibrous thickening and condensation of the tissue. The results of arteritis are: (1) Degeneration and atheroma; (2) constriction and even obliteration of the arterial channel, which occurs mainly in the small and medium-sized arteries; (3) dilatation, either general or irregularly distributed, or purely local. In the latter case it constitutes aneurysm.

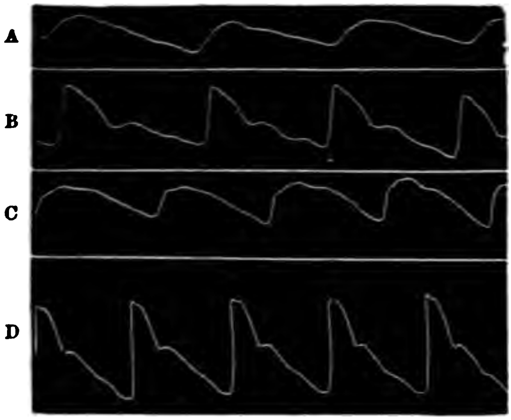
Symptoms.—Acute aortitis is probably not revealed by symptoms; but cases of acute arteritis have been recorded affecting the vessels of the extremities. In one such case pain and tenderness, limited to the course of the vessels, gradually spread down the limbs to the extremities. In others there have been pain, tenderness, loss of pulse in the affected vessels, gangrene of portions of skin in the area corresponding to their distribution, or shedding of the nails. General arteritis has also been seen in a chronic form, leading again to pains in the limbs along the course of the arteries, and followed by obliteration or such narrowing of the channel as to abolish the pulse.

ARTERIAL DEGENERATIONS.

Arterial degeneration is common in the latter half of life, and is known under the names of *arterio-sclerosis* and *atheroma*. It occurs as a senile change, without other appreciable cause; it arises from various chronic intoxications, as those of alcohol, lead, gout, and syphilis; from excessive strains of manual or other labour; and in connection with Bright's disease, of which it is perhaps more often the antecedent than the sequel.

As a senile change it is probably of purely degenerative nature, causing a fatty change in the cells of the innermost layers of the intima; but in some other instances the process follows upon definite inflammatory changes such as have just been described. Thus the grey semi-cartilaginous patches become mixed with others of a yellow colour, which arise from a fatty change taking place in the inflamed tissue. A pasty mass results, which contains fat granules and cholesterin crystals. It affects chiefly the deeper layers of the inner coat, but may approach to the surface, so that the innermost layers finally give way, and an atheromatous ulcer results. In some cases calcareous granules are deposited and form plates or spicules, upon which fibrin may be deposited from the circulating blood. The combination of patches of early inflammation, atheroma and calcareous deposits, with the irregular dilatation of the vessel which occurs as the various weakened spots yield to the blood-pressure, constitutes *endarteritis deformans*. In the senile forms of arterio-sclerosis the arteries are dilated, tortuous, and rigid. There is extreme degeneration of the media, and calcareous plates are deposited on the intima.

FIG. 29.



A. Right Radial Pulse in a Case of Aortic Aneurysm compressing the Right Innominate Artery.
B. Left Radial Pulse in the same Case.
C. Radial Pulse in Compression of the Subclavian Artery.
D. Atheromatous Artery. Pressure, Six Ounces.

Symptoms.—When at all extensive, atheromatous degeneration can be recognised by its effects upon those arteries which are accessible to examination during life. First, the artery-wall becomes rigid; secondly, yielding to the pressure of the blood-current the walls become stretched, and make the vessel both larger in its tranverse diameter, and at the same time longer; thirdly, the increased length is accommodated by the vessel becoming tortuous instead of nearly straight. At each beat of the heart the artery is not only expanded, but bent still more out of a straight course. To the finger the artery feels hard, rigid, and rough, whilst the pulse consists of a sudden expansion, and a slow almost unbroken collapse. Fig. 29, D, shows the characters of a sphygmographic tracing.

It can hardly be said that any general condition of illness necessarily accompanies atheromatous arteries. Many persons in advanced life with this form of degeneration are in the enjoyment of perfect health. But such diseased vessels frequently co-exist with chronic Bright's disease; by their want of elastic tissue they tend to hypertrophy and dilatation of the heart; and they lead to serious results in several ways, by rupture (cerebral hæmorrhage), by thrombosis and obstruction (hemiplegia, gangrene of the limbs), or by local dilatation (aneurysm). Thus while itself often an indication of senility of tissues, arterio-sclerosis and atheroma should forewarn us as to possible accidents.

The *lardaceous* or *amyloid* change is a form of degeneration affecting vessels, of which more will be said under Lardaceous Disease of the Liver.

ANEURYSM.

This name is applied to dilatation of an artery for a more or less limited extent of its course. Aneurysms have been divided, according to their shape, into *fusiform* and *sacculated;* the fusiform being a more or less uniform dilatation of the whole circumference of the vessel; the sacculated forming a globular projection from one side of the vessel, and connected with it in advanced cases by a constriction or neck. They have been divided into *true* and *false*, according to the number of the arterial coats still present in the sac; but it appears that the inner coat never persists in aneurysms above a certain size, and hence this distinction has no apparent value. Sometimes, especially in the limbs or the abdomen, a sacculated aneurysm ruptures at a prominent point, blood oozes slowly out into the tissue around and forms a coagulum, bounded by a kind of cyst of inflammatory tissue. This has been called a *diffused* aneurysm. Lastly, a *dissecting* aneurysm is formed when at a part of the artery affected with atheroma the blood penetrates the inner and middle coats, and forces its way outside these two, between them and the outer coat.

Ætiology.—Aneurysms arise from any cause that weakens the vessel at one point. The most common cause is atheroma, especially in the large vessels, in which the inner and middle coats are weakened, and the whole wall yields to the pressure of the blood at that point. In smaller vessels—for instance, those of the brain and lungs—the vessel may be weakened by the local causes of arteritis already spoken of—viz., embolism, or the invasion of tubercle. Surgical injuries of the outer coat also lead to aneurysm. Of the more general causes predisposing to aneurysm, syphilis holds an important place; and probably also excessive muscular strain acting through the circulation.

Results.—These are mostly seen in the sacculated forms. One result is the *coagulation* of blood in the sac itself. As this is out of the direct current it moves more slowly, and has a tendency to coagulate. The sac is thus frequently lined, or nearly filled with successive layers of pale buff, fibrinous deposits: and it is by the complete filling of the sac with these fibrinous layers that aneurysms are mostly obliterated and cured. The greater the freedom of communication with the main vessel the less the liability to the formation of fibrin, and in the case of a fusiform aneurysm no deposits take place.

Another result of aneurysm is its *pressure* upon the parts around it. The sac may attain an enormous size; an aneurysm, of which there is a model in the Museum of Guy's Hospital, springing from the aortic arch, measured eight inches in diameter. As it enlarges, the growth presses with irresistible force upon adjacent parts, displacing the various organs, compressing and obstructing the blood-vessels, the trachea, the bronchi, or the œsophagus, flattening and stretching nerves, and causing thereby pain, numbness, or paralysis, according to the nerve involved, and the degree of its compression. When it comes in contact with unyielding bone, an aneurysm causes absorption of the osseous tissue, and excavates or actually perforates it. The vertebræ are frequently eroded in this way, and it is remarkable that the intervertebral cartilages are more resistant than the bone, so that when the aneurysm is large enough to cover more than one vertebra, the cartilage projects between the two cavities which have been made in the adjacent bones. The ribs and sternum, at first slightly raised by the advance of an aneurysm, are subsequently perforated, and allow the pulsation of the tumour directly under the skin. Analogous results occur in other parts of the body, but it is chiefly in the thorax that the pressure-effects of aneurysm are manifested, because the bony walls allow no room for their important contents to escape.

A third effect of aneurysm is *hæmorrhage*, which is the cause of death in a large percentage of cases. The great distension of the coats, and the degeneration which precedes this, sufficiently explain why hæmorrhage occurs. Even the deposit of layers of fibrin within the sac will not always prevent it; the clot, if at all abundant, does not organise, and the blood may force its way into fissures and meshes of the coagulum, and so finally reach the surface, and slowly ooze out. The rapidity and extent of the hæmorrhage further depend upon the support the aneurysm has from without. Ruptures into hollow viscera and serous cavities are often rapidly fatal; though I have known a man live ten days after a rupture into the pleura. Ruptures into connective tissue or inter-muscular spaces are often much slower, and in the limbs may allow time for successful treatment.

Symptoms.—They may be divided into those common to all aneurysms, and those determined by the locality; of the latter only the special symptoms of thoracic aneurysm will be now considered, those characteristic of abdominal aneurysm being discussed elsewhere (*see* Abdominal Tumours).

The symptoms common to aneurysm in any part of the body are:—(1) Tumour; (2) Pulsation; (3) Murmur; (4) Pain; and (5) Other effects of pressure.

Tumour, or some kind of swelling, is a necessary part of an aneurysm, but it may, of course, be entirely unrecognisable during life in such parts as the cranium, the thorax, the deeper parts of the abdomen, or the gluteal region.

Pulsation is the characteristic symptom, showing the connection of the tumour with an artery. It is synchronous with the cardiac systole and the radial pulse, mostly rather slow and heaving, expansile—that is, enlarging the tumour in all directions at once, and not merely in a direction perpendicular to the course of the artery apparently affected, which would be the kind of pulsation communicated by an artery to an independent tumour over it. Pulsation is affected by the amount of coagulum lining the sac, and is in some cases absent.

Murmur.—On listening with the stethoscope over an aneurysm in the abdomen or limbs a murmur is mostly audible, synchronous with the beat of the pulse, varying in quality, soft, harsh, or loud. It is due to the passage of the blood from the normal artery into the wide cavity of the sac. In some cases a diastolic murmur is present; and with a feeble current of blood, or with much coagulum in the sac, and perhaps under other circumstances, the murmur may be absent.

Pain is frequently present, and results from the stretching of, or pressure on, parts in the neighbourhood. It occurs at the seat of the aneurysm, and often radiates in different directions from it—to the shoulder and down the arm, for instance, in thoracic aneurysm, and along the course of the intercostal nerves when the spine is eroded.

The other effects of pressure will vary with the seat of the aneurysm.

Aneurysm of the Thoracic Aorta.

Aneurysm may occur at any part of the thoracic aorta, from the sigmoid valves to the diaphragm; but the first part is by far the most frequently involved, and here especially all varieties occur, from irregular dilatations of the whole calibre to true sacculated aneurysms.

The results of the former, *dilated aorta*, are not seen so much in the pressure effects. If the part adjacent to the valves is affected, the orifice may be dilated, the valves are thus rendered incompetent to close it, and regurgitation will take place, followed by hypertrophy and dilatation of the left ventricle. The symptoms are chiefly those of aortic regurgitation. In other cases of dilated aorta there is a systolic murmur, and the patient suffers from attacks of severe cardiac pain, with many of the characteristics of *angina pectoris*, on anything beyond the most moderate exertion. Such patients sometimes die suddenly.

A *sacculated aneurysm* causes symptoms which depend on its position and the direction of its growth. In the first part of the arch it may grow forwards and present itself as a pulsating tumour in the second or third right intercostal space, less commonly in the second or third left space. The tumour is slightly tender, often the seat of pain, which is aggravated by exertion; and commonly a soft systolic murmur is heard over it. Growing towards the right, a tumour in this region presses upon the superior vena cava, causing œdema of the upper extremities, or it grows into the upper part of the right chest, compressing the upper lobe of the right lung, or the bronchus leading to it, and producing dulness and deficient breath-sound over the corresponding area. Towards the left an aneurysm may press upon the pulmonary artery, cause dilatation of the right heart, and ultimately open into the pulmonary artery. Rupture into the pericardium is a not infrequent termination of aneurysms in this situation.

In the second part of the arch, an aneurysm especially affects the convex border at the site of the origin of the great vessels, and frequently grows upwards to the base of the neck, where it forms a pulsating tumour, and is with difficulty distinguished from aneurysm of the carotid or innominate artery. The pressure effects, if present, are mainly directed towards the trachea, producing stridulous breathing and dyspnœa; while the tumour itself is indicated by dulness over the upper end of the sternum, and by murmur. Aneurysms arising from the concave part of the arch come into relation with the left bronchus, which passes under it, and the left recurrent laryngeal nerve, which winds round it. Pressure on the bronchus obstructs the entrance of air into the left lung, and is indicated by deficient respiratory murmur over the left chest, while the percussion-resonance is retained. Another result of pressure on the bronchus is that when, with the patient in the upright position, his mouth closed, and his chin raised to its fullest extent, the cricoid cartilage is grasped by the finger and thumb and gently lifted, the pulsation of the aneurysm is conveyed to the fingers holding the cricoid (W. S. Oliver). This sign, called "*tracheal tugging*," is said by Dr. MacDonnell, of Montreal, to be only produced by aneurysm of the transverse arch; and although further observations have shown that slight tugging can be felt in some healthy persons, it is a valuable indication when very well marked. Pressure on the recurrent laryngeal nerve causes abductor paralysis of the left vocal cord, with subsequent "paralytic contracture" of the adductor, so that the cord occupies the middle of the glottis, and a certain amount of stridor, and clanging cough may be the result.

Aneurysms of the aorta below the arch cause, according to their position, dysphagia from pressure on the œsophagus, severe pain from erosion of the spine, or pressure upon the intercostal nerves; in more advanced cases, paraplegia from implication of the spinal cord. A murmur is often heard over the spine behind under these circumstances. Compression of the bronchus is at first indicated by diminution of the respiratory murmur. Later on it causes collapse of the lung, and dilatation of the bronchial tubes, with partial retention of secretion, giving dulness, bronchial or cavernous breathing, and moist râles as physical signs (*see* pp. 425, 430). Compression of the lung itself may occur, with localised dulness and loss of respiratory murmur; and in some cases gangrene.

Some other symptoms may be mentioned which are common to two or three of the situations discussed. *Inequality of the pupils* occurs from interference with the sympathetic. Mostly the pupil on the affected side is contracted from paralysis of sympathetic fibres: but it may be the larger; and in some cases the pupil of the affected side is at one time larger, at another smaller. *Inequality of the radial pulses* is another important indication of thoracic aneurysm. The pulse on one side is smaller, less sharp in its rise, and shows in the sphygmographic tracing a sloping upstroke, or even complete absence of the percussion wave (Fig. 29, A, B, C). This results either from the aneurysm compressing the innominate artery, or the subclavian, according to the side affected; or from the obstruction of the origin of one of these vessels by coagulum formed within the aneurysm.

Loss of flesh, cough, and dyspnœa on exertion or in paroxysms, are the common accompaniments of thoracic aneurysm of any size. Death takes place by exhaustion from pain and malnutrition; by interference with necessary functions from pressure upon such parts as the œsophagus, trachea, or bronchus; by inflammatory and septic processes from pressure on the lung; and, lastly, by rupture of the sac and hæmorrhage.

Diagnosis.—The diagnosis must be made from a careful consideration of the above facts, remembering that the symptoms vary with the seat of aneurysm. Some of the combinations which may give rise to suspicion of aneurysm may be enumerated:—

(*a*) Pain of angina-like character, with systolic murmur over the base of the aorta; (*b*) systolic murmur occurring at a part of the chest not corresponding to the seat of any valvular orifice; (*c*) pulsating tumour in or near the præcordial region; the points at which a normal or enlarged heart may beat must be remembered, and especially it must not be forgotten that pulsation of the top of the right ventricle in the second left interspace is quite common; (*d*) obstruction of one or other radial pulse; (*e*) obstruction of veins; (*f*) obstruction of trachea, bronchus, or œsophagus; (*g*) paralysis of one vocal cord, especially the left.

Aneurysms near the base of the aorta are more likely to be confounded with valvular disease; those in the deeper parts of the chest with new growth (*see* Mediastinal New Growths). The difference has been stated in another way: namely, that aneurysms of the first part of the arch cause more physical signs, those of the transverse arch, more symptoms. The extremely rare occurrence of a pulsating pleurisy or empyema has been referred to (*see* p. 481).

Prognosis.—This is very unfavourable in the case of thoracic aneurysms. Complete consolidation with cure is rare; relief of symptoms and postponement of the fatal result for some years may be often effected.

Treatment.—The objects of treatment are to lessen enlargement, to prevent rupture, and to favour coagulation of blood in the sac. Everything which will quiet the circulation and diminish the force of the heart's contraction will act favourably in these directions. The surgical methods employed in aneurysms of the limbs are rarely applicable in the case of thoracic aneurysms. Ligature of the vessel on the cardiac side of the sac is out of the question, and distal ligature or compression can only be applied to branches such as the carotid and subclavian. Insertion of horsehair, wire, or needles, and the injection of astringent fluids into the sac have had too little success in aneurysms of the limbs, where the operation is more under control, to be recommended for aortic aneurysms. Galvano-puncture has, perhaps, been more successful. The process consists in the introduction of two needles, connected with the poles of a battery, into the sac of the aneurysm; as a result of the passage of the current fibrin is deposited on the poles, but the danger of portions being detached, and causing embolism in remote parts, is present here, as it is in the methods just mentioned. The treatment of aneurysm in the chest generally resolves itself into: (1) rest; (2) restricted diet; (3) the use of anodynes and sedatives; (4) iodide of potassium. Rest must be absolute, the patient being in the recumbent position, and not allowed to sit or stand up for any reason whatever. The diet which was recommended by Tufnell, who strongly advocated this plan of treatment, consisted per diem of ten ounces of solid, including three of meat, and eight ounces of fluid, divided into three meals. Opium or morphia is generally given in some form to ease pain, to produce sleep, or calm restlessness, but other sedatives may also be of use, such as bromide of potassium, chloral, paraldehyd or sulphonal. Pain may also be relieved by belladonna applications, or by cold; or by venesection to a small amount. Iodide of potassium appears to have a special influence upon the coagulation of blood in aneurysms, as great improvement in diminution of pulsation and of pain has followed its use, even though unassisted by restriction of diet. It should be given in increasing doses, up to 60, 90, or 100 grains daily, so long as the quantity given has a slowing, and not a quickening effect upon the pulse. Where the vocal cords are implicated by pressure on the recurrent laryngeal nerve, tracheotomy may be required to prevent fatal asphyxia. But aneurysms also cause dyspnœa by tracheal compression, for which tracheotomy would afford no relief.

PHLEBITIS.

Inflammation of the veins, or phlebitis, results in thickening and infiltration of the walls with leucocytes, which may be in such numbers as to constitute a real suppuration of the coats. The terms *endophlebitis* and *periphlebitis* have been used to indicate inflammation of the intima and the adventitia respectively. Periphlebitis arises from contact with inflammatory foci outside the vein, or from injury. Endophlebitis is most often set up as a result of thrombosis, or coagulation of blood in the vein itself. This occurs from a variety of causes (*see* Thrombosis). The clot may then adhere to the vein-wall, becoming at the same time organised, and the vein may be completely obliterated. On the other hand, the clot may become channelled, and allow the continuation of the circulation; or in other cases it softens down into a puriform fluid. Periphlebitis extending inwards itself leads to thrombosis; on the other hand, abscesses may form in the tissue around the vein.

Symptoms.—Phlebitis is accompanied by pain and tenderness in the course of the affected vessel, with some reddening of the surface in the case of superficial veins. The vein can be felt as a prominent hard cord, and a varying amount of febrile reaction accompanies the local disease. The formation of abscesses will, of course, be indicated by hardening of the surrounding tissue, redness, and œdema of the skin, and subsequently fluctuation. The secondary effects which result from breaking down, and transportation of the particles of thrombus, are described below.

The **Treatment** of phlebitis consists in complete rest of the part affected, the application of warm fomentations to ease pain, and the administration of opiates, if necessary, for the same purpose. If abscesses are forming, poultices should be applied, and pus may have to be evacuated by incision.

THROMBOSIS AND EMBOLISM.

Thrombosis is the name applied to the coagulation of blood within living vessels, whether arteries or veins, the clot itself being called a *thrombus*.

Embolism means the transference of a portion of clot from one part of the circulation to another, and its impaction when it arrives at a vessel too narrow for its further progress. This can only take place in the arteries (and portal veins), since the flow of blood in the veins is from those of a smaller to those of a larger calibre. The transferred particle is called an *embolus;* and this may be not only a detached portion of thrombus, but other bodies, such as particles of new growth, and minute hydatid cysts.

Thrombosis occurs from slowness of the current of the blood, or from irregularities on the inner surface of the blood-vessels or cavities of the heart; or from these two conditions combined. Blood thus coagulates in the heart upon its inflamed valves, as has been already described. It coagulates in the arteries, chiefly in smaller vessels, the calibre of which has been made uneven by atheroma, and in aneurysmal dilatations; and, lastly, it coagulates in veins, in which the current is normally much slower than elsewhere, and may fall, from local and general causes, to an extreme point. The first effect of the coagulation of blood in a vessel is naturally to cause an obstruction, which will have different effects according as it is in an artery or in a vein. The coagulation once formed receives further deposits of fibrin from the blood circulating above and below, and so the thrombus may extend into larger and larger vessels. When first formed it is soft, and fills the veins; but after a time it may shrink, and thus allow the re-establishment of the circulation. But the termination is not always so favourable. The thrombus commonly sets up some endarteritis or endophlebitis, adhesion to the wall of the vessel takes place, and ultimately the clot becomes organised, with permanent obliteration of the channel. In other cases septic micro-organisms may cause the coagulum to break down into a puriform fluid, which consists of pus-corpuscles, micrococci, and fine granular particles. An important result of thrombosis in the heart and in the veins is the detachment of fragments from the coagulum, and their transference to other parts of the circulation. They then become *emboli*, as above stated. The results differ according to the position and character of the original thrombus. Portions detached from venous thrombi are carried by the current of blood into the right auricle, thence into the right ventricle, and into the pulmonary artery, which they may block according to their size, either quite at its commencement, or in the substance of the lung. Thrombi in the right side of the heart will similarly cause embolism of the pulmonary artery; but thrombi on the aortic or mitral valves will cause embolism of the systemic arteries in the brain, spleen, kidneys, limbs, or elsewhere.

The results of embolism are somewhat peculiar. When a small particle of clot is impacted in a vessel of the kidney, or spleen, the first effect seems to be that the more or less wedge-shaped or conical area of tissue supplied by the vessel becomes turgid with blood, which is actually extravasated into the tissue, forming a conical plug with its apex at the point of obstruction, and its base at the surface of the organ. This mass (known as an *infarct*, or hæmorrhagic infarct) gradually becomes decolourised; and in a later stage has a whitish-yellow tint, with a zone of red around it. Ultimately it becomes uniformly pale yellow, and shrinks in size, so as to form a depression on the surface; and it may remain thus for long periods. But if the embolus comes from a suppurating thrombus, or is the product of malignant endocarditis, then it sets up similar suppurative or septic processes in the part to which it is carried, as a result of the micrococci carried with it. The infarcts then tend to become purulent in the centre, and to form actual abscesses, such as occur in the lungs in pyæmia; or cavities with puriform contents, like those seen in the brain and kidney occasionally in malignant endocarditis.

The following are the more usually recognised forms of thrombosis and embolism:—

Femoral Thrombosis.—This arises in the last stage of phthisis, cancer, and other exhausting diseases, in convalescence from typhoid fever, and after confinement (phlegmasia alba dolens). The leg becomes swollen, and the vein can be felt to be obstructed; there is generally also some tenderness from co-existing phlebitis. The detachment of a portion of clot followed by its impaction in a large branch of the pulmonary artery with sudden death, is an occasional accident.

Jugular thrombosis, and thrombosis of the *lateral sinus*, result from disease of the internal ear, or mastoid cells. From contact with the external ear, septic organisms are frequently present, severe phlebitis is set up, and the clot becomes septic. Particles are then conveyed through the right side of the heart to the lungs, in which pyæmic abscesses are formed.

Thrombosis of the *pelvic veins* arises from disease of the pelvic viscera in women, or from gonorrhœa in both sexes.

Large clots sometimes form in the heart, just previous to death, when the circulation is failing, and in recesses of the walls in cases of dilatation. They may hasten death by hampering the action of the organ, or they may supply emboli to the pulmonary or systemic circulation.

Embolism and thrombosis of the *cerebral arteries* are described under Diseases of the Brain (p. 285).

Embolism of a large artery in a *limb* is not a very common event. It causes sudden acute pain, followed at once by numbness, coldness, and loss of power in the limb: the pulse is imperceptible below the seat of embolism, and gangrene sometimes results. In the *spleen* and *kidneys* the occurrence of embolism is not so commonly recognised. Sometimes there is sharp pain in the left side from embolism of the spleen. Embolism of the kidney causes frequently albuminuria, with perhaps blood in the urine; and in malignant endocarditis there is often double nephritis, probably from minute emboli (*see* p. 505). Cases of embolism of the *mesenteric artery* have occurred in which the patient has been seized with severe abdominal pain and distension, followed by collapse and death in one or two days; and blood has been found in the bowel and in the peritoneal cavity. Very similar results may follow thrombosis of this artery, but the symptoms are more slowly developed.

The effects of embolism and thrombosis of the vessels of the *liver* are described under Pylephlebitis.

In the *pulmonary* circulation, embolism of the largest trunks may occur, commonly as a sequel to femoral thrombosis, when death is often quite sudden. At other times, the event is signalised by sudden collapse, sense of suffocation, and urgent dyspnœa; of which, though generally fatal, cases of recovery have been recorded. Pulmonary infarcts are mostly the result of embolism of the smaller vessels. They are the conical hæmorrhagic masses which are seen in the lungs in chronic heart disease, and arise by embolism from coagula in the right cavities of the heart. Their occurrence often gives rise to hæmoptysis, and if they are large, there may be dulness and deficient respiratory murmur at the surface of the chest corresponding to them. Pyæmic infarcts of the lung have been already described (*see* p. 143).

FUNCTIONAL DISORDERS OF THE VASCULAR SYSTEM.

Excessive Pulsation of the Abdominal Aorta.

This is often mistaken for abdominal aneurysm. It is more frequent in women than men, and occurs between the ages of twenty and forty-five. The patients are generally nervous, hysterical, or hypochondriacal; and it is often associated with some dyspeptic symptoms. There is constant complaint of pain, distress, and throbbing over the abdominal aorta, which can be felt and seen beating with unusual force. If it be carefully examined, it will be found that its outline is cylindrical like that of the normal vessel, and that there is no fusiform or saccular enlargement. Firm pressure with the stethoscope may elicit a slight murmur, but usually there is no more than the dull sound of impact of the vessel against the instrument. The rest of the circulatory system is normal. The trouble may continue for months or years, without any material alteration.

Treatment.—Gastric disorders should be met by appropriate methods, and the bowels should be kept open. Exercise, fresh air, the avoidance of sedentary occupations, or of the chance of brooding over the complaint, should be enjoined; and, medicinally, bromide of potassium in full doses should be tried.

Raynaud's Disease.

(*Symmetrical Gangrene.*)

This disorder, first described by Dr. M. Raynaud in 1862, as local asphyxia and symmetrical gangrene of the extremities, is due to a spasmodic contraction of the arterioles, whereby the circulation in the affected parts is retarded, so as to cause a temporary " deadness " or lividity of the part, or is obstructed to such an extent or for such a long period as to be followed by actual gangrene.

Ætiology.—It is much more frequent in women than in men, and is first noticed commonly between the ages of fifteen and thirty, or even in childhood. Many patients are delicate, or anæmic, nervous or hysterical, but some seem to have been in good health until the occurrence of the disease. Hæmoglobinuria, peripheral neuritis, various skin eruptions, and, rarely, ague, have occurred in association with the symptoms of Raynaud's disease. Cold and emotional disturbance are exciting causes.

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resemblance to local asphyxia, and perhaps may have an allied pathology; they occur from definite exposure to cold.

Prognosis.—Many cases recover. Death is rare as a direct result of the gangrene.

Treatment.—Raynaud obtained good results, at least in cases of less degree, by the application of a continuous galvanic current, with the anode at the back of the neck, and the kathode over the sacrum and lower lumbar region. Barlow recommends that the affected limb should be immersed in a basin of salt and water, and that one pole of the battery should be applied to the top of the limb, and the other placed in the water. A current should be used as strong as the patient can bear. Shampooing the limb and diffusible stimulants internally may also be employed. Cold and excitement should be avoided.

ERYTHROMELALGIA.

This is the name given by Weir Mitchell to a complaint in which there is acute pain in the feet and legs, associated with dilatation of the vessels. The pain is acute, burning, occurring in paroxysms, aggravated by heat and exercise, and relieved by rest and cold. When the patient stands or walks, the parts become flushed or dusky red. By some it is regarded as a neuralgia, or neuritis; by others as chiefly a vasomotor failing, and allied to Raynaud's disease; by others again as probably dependent on changes in the spinal cord, such as occur, for instance, in syringomyelia. Two of Mitchell's cases subsequently developed spinal symptoms. The suggestion has been made that some cases of erythromelalgia are due to ergotism.

ANGEIO-NEUROTIC ŒDEMA.

This is another curious affection apparently connected with the vasomotor apparatus. Circumscribed swellings appear on various parts of the body, for instance, on the face, the eyelids, the hands or legs, or in the throat. They are not inflammatory, and not dependent upon gravity; they are accompanied by burning, pricking, and itching. They appear suddenly, are of short duration, but recur frequently and even daily; they are generally harmless, but œdema of the larynx has sometimes proved fatal. Gastro-intestinal symptoms are usually present, such as colic, nausea, and vomiting. The disease is often hereditary, and very intractable.

DISEASES OF THE MEDIASTINUM.

MEDIASTINITIS.

Inflammation of the mediastinum may be suppurative or non-suppurative. The former, or *mediastinal abscess*, arises from numerous causes, of which injuries by bullet, stab, or blow, and tuberculosis of the lymphatic glands are the most frequent, while occasionally it follows upon pneumonia, pleurisy, erysipelas, or enteric fever. The abscess may be in the anterior or in the posterior mediastinum, more often in the former. The chief symptoms are sternal pain and pyrexia. Physical signs will only be apparent if the abscess reaches a sufficient size, when there may be dulness, localised tenderness, œdema over the sternum, and ultimately perhaps fluctuation at the border of the sternum. The pus must be evacuated as soon as possible, and, if necessary, the sternum must be trephined or resected.

Adhesive, simple, or non-suppurative mediastinitis has been less often recognised than the suppurative form. It may also arise from traumatism and general infectious diseases, but its most common associations are pleurisy and pericarditis, especially the latter, forming then a *mediastino-pericarditis*. Under these circumstances the tissues of the anterior mediastinum may be matted together to form dense fibrous tissue, and with this there is pericardial adhesion or thickening, and sometimes pleural adhesion or fluid. It is very doubtful if indurative mediastinitis can be recognised apart from the chronic pericarditis with which it is associated, and the symptoms which occur are generally the dropsy, dyspnœa, enlarged liver, and ascites, which would be explained by the hypertrophy and dilatation associated with adherent pericardium. If from any such combination of symptoms, with absence of the physical signs of valvular disease, an adherent pericardium can be diagnosed, the co-existence of past or present pleurisy would make mediastinitis probable. At the same time its importance seems somewhat small by the side of the immediate cardiac lesions.

TUBERCULOSIS OF THE BRONCHIAL GLANDS.

(*Bronchial Phthisis.*)

Ætiology and Pathology.—The former is similar to that of tubercular infection in general; and the bronchial glands are infected commonly from the lesions of pulmonary phthisis. But there is this occasional point of difference, that in children the glands may caseate, enlarge, and even suppurate with very little or no evidence that the lungs are involved; and these are especially the cases to which the name of bronchial phthisis has been given. The bronchial glands go through the same changes as other organs when invaded by tubercle. Grey granules are at first formed, and caseation follows; the glands become enlarged, and may suppurate, or become calcareous. As a result of their enlargement they press upon surrounding parts, especially the œsophagus and the trachea or main bronchi. If they suppurate they may discharge into one of these passages, or they may lead to abscess in the mediastinum.

Symptoms.—Enlarged bronchial glands constitute a mediastinal tumour. They cause pain in the chest, cough, and dyspnœa, and perhaps slight swelling of the face and neck, dysphagia, and hæmoptysis. The cough is of most interest. It is sometimes harsh and altered in quality, as if the larynx were involved; and this is probably from some pressure of the glands on the recurrent laryngeal nerves, producing paresis of one or both cords. The cough may be constantly hacking, or spasmodic like that of pertussis. If the pressure on the trachea is considerable, there is severe dyspnœa with stridor, and impending suffocation; and this is especially likely to be the case if the glands are suppurating and rupture is imminent. When rupture takes place, pus is expectorated, and there is some danger of asphyxia in young children. Occasionally a detached fragment of gland has been impacted in the respiratory passages, and caused death. The puffy swelling of the face and neck is due to the pressure on the superior vena cava or one or other innominate vein. There is more or less pyrexia, according to the stage and the rapidity of the caseating process.

Physical Signs.—If the glandular mass is of great size, it will cause dulness in the back between the scapula and the spine on one or both sides; and more rarely over the upper part of the sternum in front. The auscultatory signs are variable, even independent of any co-existing phthisis. There may be deficiency of breath-sounds over the lung on one or other side; or bronchial breathing and bronchophony in the neighbourhood of the enlarged glands.

Diagnosis.—The symptoms and physical signs of what is practically a mediastinal tumour in a young child, with a family history or other evidence of tubercle, will generally enable the disease to be recognised; but in a small child it may be easily confounded with empyema, until tested by exploration.

The **Prognosis** is not absolutely unfavourable. Health may be restored completely after the discharge of pus; and other cases, no doubt, get well on calcification of the tubercle.

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vena cava, and upwards in obstruction of the inferior cava. This last, however, rarely results from mediastinal tumour, although it is possible for a malignant growth to reach the inferior cava, just above the diaphragm.

Arteries often maintain their course through a tumour unmolested. They are sometimes compressed, with the effect of weakening or obliterating the peripheral pulse.

The results of *compression of the trachea and bronchi* have been already described (pp. 412, 430). If the *œsophagus* is involved there will be difficulty in swallowing, and an œsophageal bougie will meet with resistance.

The paralyses of the vocal cords, which follow pressure on the *recurrent laryngeal nerves*, are similar to those caused by aneurysm. The left nerve is more exposed than the right from its lower position, and the results will be abductor paralysis, or complete paralysis, according to the degree of compression. Sometimes there are spasmodic attacks of dyspnœa.

The *displacement* of *organs* and *deformity of the chest* depend on the size and position of the tumour. The heart may be pushed to the right or left, the liver downwards towards the abdomen, or one lung may be compressed against the ribs. Deformity may be quite absent, especially if the tumour arises in the posterior mediastinum. But there may be enlargement of one side from effusion of fluid into the pleura, caused by the growth pressing on the veins in the root of the lung; or retraction of one side if the growth compresses the bronchus. Tumours in the upper part of the chest may extend into the root of the neck, or be accompanied by enlargement of cervical glands in this situation. Dyspnœa, spasmodic cough, expectoration of mucus, hæmoptysis, faintness, attacks of syncope, and palpitation are additional symptoms in many cases. Paraplegia occurs in exceptional cases from invasion of the spine, and cachexia may be pronounced when the symptoms are prolonged and severe.

Physical Signs.—The most important is the decided dulness which a tumour produces if it reaches the surface of the chest, or if it compresses the lung between itself and the chest-wall; but a considerable tumour may exist in front of the spine without appreciably altering the signs of percussion. The auscultatory sounds will depend on the presence or absence of fluid in the pleura, and on the relation of the tumour to the root of the lung (*see* p. 430).

Diagnosis.—An *aortic aneurysm* is most readily confounded with mediastinal new growth. An aneurysm is itself, of course, a mediastinal tumour, occupying the middle or superior mediastinum when it grows from the arch, the posterior mediastinum when it arises from the thoracic aorta. A diagnosis is, nevertheless, very desirable, since the prognosis as to duration of life and temporary relief of symptoms is much more favourable in the case of aneurysm. Many of the symptoms are necessarily the same: they both press upon and displace important structures, such as the œsophagus, the trachea or bronchi, and the large nerve-trunks. One of the most important distinctions is the fact that aneurysms implicate arteries, and thus may lead to suppression of pulse on one or other side; while new growths commonly spare the arteries, but compress and project into the veins, producing venous distension and œdema. The presence of a murmur, and the duration of the symptoms for more than twelve or fifteen months, are in favour of aneurysm. Enlargement of the cervical glands, or the existence of tumour in other parts of the body, speaks for new growth; in this connection the mammæ or the testes should always be examined. The stridor produced by compression of a bronchus should not be mistaken for the rhonchus of *bronchitis*. The former is constant in time and position; the latter is variable, and changes from place to place within a few hours, or disappears at intervals.

The **Prognosis** is bad, and the duration rarely more than a few months.

Treatment.—This is mainly palliative, and very little can be done beyond relieving pain by opium or morphia. Symptoms threatening life, such as laryngeal asphyxia, must be met as they arise.

DISEASES OF THE ORGANS OF DIGESTION.

PHYSICAL EXAMINATION OF THE ABDOMEN.

THE abdomen is accessible to the same methods of examination as are employed in the case of the lungs and heart—namely, inspection, palpation, mensuration, percussion, and auscultation; and in most instances it is desirable that the patient should be in the recumbent position, with the head rested.

INSPECTION.

The first thing to notice is the size of the abdomen. This is extremely variable even within the limits of health. It may be uniformly much enlarged; but it requires the help of other methods of examination to determine whether this is due to a collection of liquid in the peritoneal cavity (*ascites*), to gas in the intestines (*meteorism*), to fat in the parietes and omentum, or to some tumour, such as an ovarian cyst. Uniform and symmetrical retraction of the abdomen is seen in starvation, in emaciating diseases, and in death from cerebral diseases, such as tubercular meningitis and intracranial tumour.

By inspection may be observed various local enlargements or prominences, such as result from tumours or enlargements of different organs. Many of the tumours which occur in the abdomen may, under favourable circumstances, be visible on the surface; this is much more likely to occur when the patient is thin, and the abdomen empty; much less likely when the patient is fat, and the abdomen is distended with ascites or flatus. Amongst those more commonly observed are enlargements and tumours of the liver, cancer of the stomach, an abnormally dilated stomach, irregular distension of the intestines in cases of obstruction, matting of the intestine in tubercular peritonitis, enlarged spleen, hydronephrosis, the pregnant uterus, ovarian cysts, and a distended bladder. Local enlargements in the upper part of the abdomen may cause asymmetry of the thorax by driving upwards and outwards the lower ribs on one side, th enlarging the angle between the costal margin and the midd line. This is especially seen in cases of hydatid and abscess the liver.

It is important to note the relation of the abdomen to th respiratory movements, already referred to in connection with th chest (p. 379); the descent of the diaphragm is impeded by muc distension, and is checked by acute inflammation of the per toneum, so that in these cases respiration is almost entirel thoracic. In other cases respiration affects materially the positic of the organs in immediate contact with the diaphragm—namel the liver, spleen, stomach, and kidneys; whereas, organs tumours situated lower down, or connected with the posterior wa of the abdomen, are much less influenced by the descent of th muscle. The pulsations of the aorta, or of an aneurysm, or the enlarged liver in tricuspid regurgitation, and the peristalt movements of the intestines, may sometimes be seen, the latt being visible in proportion to the thinness of the abdomin parietes, and the vigour of the peristalsis.

The sudden expiratory effort of coughing will sometim produce local prominences, which are not present under ordina circumstances. These are always due to weakness or deficienci of the abdominal wall, and the projection constitutes for the tin being a hernia. Besides the familiar inguinal and umbilic herniæ, it is common to see a protrusion in the middle line fro yielding of the parietes between the two recti muscles; and occ sionally the abdominal walls, just above Poupart's ligament bulge in coughing. I have seen the same bulging just below th ribs on one side from local paralysis of the upper part of th abdominal muscles.

PALPATION.

This is undoubtedly the most important means of examini the condition of the abdomen. The abdominal walls should relaxed as much as possible, and for this purpose the patie should be in the recumbent or semi-recumbent position, and t head should be supported, for if the patient raises his head—*e.* to see what is going on—the recti abdominis become tense. T relaxation of the abdominal walls is sometimes assisted by raisi the patient's legs, but they must be supported in that position a pillow under the knees. This proceeding compels the observ to examine very much from one side, rather hampers the use the hands, and can often be dispensed with.

In many patients the abdominal muscles are persistently ten and the abdomen is palpated with difficulty. In such cases t patient should be asked to breathe deeply in and out, and shou be engaged in conversation while the hand is on the abdome or he may be asked to lift up his head from the pillow, and keep it raised for about a minute, when the muscles will become exhausted, and for a moment afterwards the abdomen may be lax enough for the purpose. If these means fail, and an examination is of the first importance, chloroform should be administered.

The examination of the abdomen should be made with great gentleness; the hands should be warm, and should be laid flat upon the surface, and care should be taken not to force the finger-tips suddenly into the abdomen, whereby the muscles are made to contract, and trustworthy results are impossible. During the movements of deep breathing, enlargements of organs or new growths, especially in the upper part of the abdomen, may be detected, which would otherwise perhaps escape recognition. When examining the sides of the abdomen, or the flanks, the observer should never neglect to employ the *bimanual* method—that is, one hand should be placed under the twelfth rib, and another on the abdomen in front; if one hand be pressed towards the other which is still, the slightest enlargement or resistance can generally be appreciated. In particular cases it may be desirable to examine the patient in the knee-elbow position.

In the normal abdomen there is scarcely any resistance to the movement of the hand in all directions. The solid organs, liver, spleen, and kidneys are almost entirely within the bony thorax; the left lobe of the liver, which lies across the epigastrium, is of small bulk, thin, and soft; the hollow viscera yield readily to the hand, and often nothing can be recognised, except, in thin people, the pulsation of the aorta or iliac vessels.

By palpation in disease we can recognise changes in shape or size of the organs, and the existence of tumours, and can obtain information on the following points:—

The condition of tenseness or relaxation of the abdominal wall, which may be local or general.

The presence of tenderness, general or local: this may be elicited directly the hand touches the abdomen, or only when deep pressure is made.

Various kinds of movement may be felt in the abdomen—the pulsations of the normal vessels, or of an aneurysm, or of the liver in heart disease; the peristaltic movement of the bowel; the movements of air in the intestine (borborygmi); the crackling or gurgling due to air and liquid, which may be felt sometimes on gentle pressure over the cæcum in enteric fever; the coarser movements or splashing of air and liquid in a dilated stomach when somewhat sudden pressure is made upon it; and the friction of inflamed peritoneal surfaces.

Under palpation also must be included two of the methods by which ascites or fluid in the peritoneal cavity may be recognised—namely, *fluctuation* and *displacement* (*see* Ascites).

MENSURATION.

By measurements we may watch and record the progress of enlargements of the abdomen from ascites or tumour, and the hepatic or splenic dulness may be exactly estimated (*see* Percussion). Sometimes mensuration may help the diagnosis of ascites from ovarian disease, by comparison of the distances from the umbilicus to the ensiform cartilage, to the pubes, and to the iliac crest (*see* Ascites); and it will confirm the evidence of inspection with regard to hepatic tumours displacing the ribs.

PERCUSSION

It is in reference to percussion especially that we must remember that the abdominal cavity extends up into the lower parts of the bony thorax. In health the abdomen is resonant over so much of the combined surfaces as corresponds to the intestines and to the stomach—that is, all the parts below the ribs, and the costal cartilages and lower ends of the ribs on the left side below the heart. It is dull over the parts which correspond to the liver and spleen—that is, for the liver, the ribs of the right side below the upper border of the sixth in front, and the eighth at the side, and for the spleen the ninth, tenth, and eleventh ribs on the left side just behind the anterior axillary line; and with very light percussion one may recognise the extension of the left lobe of the liver across the epigastrium. The relative areas of dulness and resonance may be much altered by changes in the amount of gas in the hollow viscera, and the dull areas of the liver and spleen are moved downwards in inspiration and upwards in expiration. There is, further, much difference in the quality of the percussion note over the stomach and different parts of the intestine.

Alterations in the size of the liver and spleen, or the existence of solid tumours or cysts, will give rise to new areas of dulness, and such dulness will, as a rule, be accompanied by resistance appreciable by palpation. As constant reference to these altered conditions will be made under the diseases of the different organs, it is not necessary to specify them here.

In percussion also we have another valuable method of recognising ascites, since liquid in the peritoneal cavity, unless retained by adhesions, tends always to be lower than the intestine, which contains gas; and if the patient is examined as he lies on either side and on his back in succession, the dull and resonant areas will be found to alter their positions accordingly (*see* Ascites).

AUSCULTATION.

This method of investigation has but little application in the abdomen. Friction sounds are occasionally heard over the liver and elsewhere in peritonitis; abdominal aneurysms may be accompanied by murmurs. Combined with palpation, auscultation helps us to recognise the splashing sounds in a dilated stomach; but they can be often heard some little distance from the abdomen without any assistance.

DISEASES OF THE MOUTH, TONSILS, AND PHARYNX.

STOMATITIS.

Ætiology.—First, inflammation of the mouth may be set up by chemical or mechanical irritation, such as contact with strong acids or alkalies, or the presence of broken or carious teeth; secondly, inflammation may spread from neighbouring parts, such as the nose or naso-pharynx, so as to involve the mouth; and thirdly, special forms of stomatitis occur in some constitutional diseases, such as measles, variola, syphilis, and scurvy. Mercurial poison also leads to severe stomatitis.

Symptoms.—These are swelling and increased redness of the mucous membranes of the gums, lips, and cheeks, swelling of the tongue, salivation and increased secretion of buccal mucus, which adheres as a coating to the surface, and swelling of the neighbouring lymphatic glands. In later stages abrasion and superficial ulceration take place.

Besides the special forms due to the infectious diseases, and scurvy, there are the varieties known as aphthous stomatitis, ulcerative stomatitis, gangrenous stomatitis, and parasitic stomatitis.

APHTHOUS STOMATITIS

Occurs in children, especially about the time of the first dentition, and less frequently in adults; it consists in the formation of circular grey patches, or aphthæ, on the gums, tongue, and the inside of the lips and cheeks. They are from 3 to 5 mm. in diameter, slightly raised from the surface, and, though looking like vesicles, are caused by a fibrinous exudation beneath the epithelium. After a time the epithelium is shed, and small ash-

Treatment.—We should aim at improving the general condition of the patient. In infants the food must be rendered suitable, and the diarrhœa checked. After every meal the mouth should be carefully wiped out with a fresh piece of soft linen; and the patches should be touched with a solution of borax, or a little glycerine of borax should be left in the mouth.

PAROTITIS.

Primary, or *specific parotitis*, or mumps, has been already described amongst the infectious diseases (p. 75).

Secondary parotitis is an acute form of inflammation which arises in the course of severe illnesses, such as pyæmia, fevers, dysentery, phthisis, and carcinoma. It has been commonly regarded as metastatic, and may be so in the case of pyæmia; but in other instances it is regarded as arising from irritants, which proceed from the decomposition of matters retained in the mouth, and obtain access to the gland through Stenson's duct. Suppuration is much more common in this form; several small abscesses are formed, and afterwards run together. They may discharge externally, behind the ramus of the jaw, or burst into the external auditory meatus, or burrow deeply down the neck, or behind the pharynx. Occasionally sloughing takes place.

The **Treatment** is that of acute local inflammations; fomentations will relieve pain, and when pus is recognised, an incision must be made; but recovery depends much upon the primary illness.

Angina Ludovici, or *submaxillary cellulitis*, is a rare form of severe phlegmonous inflammation of the floor of the mouth, and upper part and front of the neck. It appears to commence in the submaxillary gland. Its treatment is mainly surgical.

CATARRHAL SORE THROAT.

Ætiology.—The causes are cold, exposure to impure air, whether in badly ventilated rooms, in hospital wards, or from open drains. The term "Hospital sore throat" expresses the frequency with which nurses and medical students are liable to these affections.

Symptoms.—In its mildest form there is only some discomfort on swallowing, and nothing may be visible in the throat. In other cases, the soft palate, uvula, pharynx, and tonsils are redder than natural, the uvula is elongated, the soft palate flaccid, and the pharyngeal veins are dilated. In more severe cases (*ulcerated sore throat*) superficial abrasions occur on the tonsils, palate, and pharynx, the tongue is furred, and there is marked constitutional disturbance. Sometimes there is excess of saliva and buccal mucus; at others there is unusual dryness of the mouth. Talking, as well as swallowing, may be painful.

Treatment.—For this we may use iron, quinine, or other tonics internally, and apply glycerine of tannic acid locally, or give rhatany lozenges. Where it is due to a hospital atmosphere, or other impure air, removal to the country for a few days may be desirable, and in other cases the improvement of the air-supply must not be neglected.

TONSILLITIS.

Inflammation of the tonsils occurs in diphtheria, scarlatina, and syphilis, as already described. In addition, we recognise a suppurative tonsillitis and follicular tonsillitis (*see also* Catarrhal Sore Throat and Chronic Pharyngitis).

Suppurative Tonsillitis.

(*Quinsy.*)

Ætiology.—This disease also arises from cold, and is most common between the ages of fifteen and twenty-five. Some persons are very liable to it, and have it repeatedly.

Symptoms.—It may affect one or both tonsils. The tonsil becomes red and swollen to twice its natural size, projecting to the middle line, and pushing the uvula aside; if both tonsils are affected they may meet in the middle line, driving the uvula forwards; the swelling and redness extend to the base of the soft palate. The surface is generally smooth, shining, and deep red or purple in colour. Externally, there is obvious swelling behind the angle of the jaw. The illness often commences with a rigor, and sickness; and the constitutional disturbance is considerable. The tongue is thickly furred, appetite is lost, and the temperature rises to 103° or 104°. Swallowing and talking are excessively painful, and saliva and mucous secretion collect in the mouth, and require to be constantly expectorated. After two to four days suppuration occurs; the tumour, which was at first hard, is now softer, and yields to the finger; or the presence of pus may be detected by placing one finger on the tonsil, and another outside behind the angle of the jaw. If the case be left, the abscess bursts into the throat, the temperature falls, and recovery quickly takes place in from four to seven days, though convalescence may be protracted for some time longer. Rarely the abscess has burrowed into the neck or chest, or eroded the carotid artery; and Fagge records a case of suffocation from entrance of the pus into the larynx.

Diagnosis.—Quinsy may resemble *follicular tonsillitis;* it is more often unilateral, the fever is more severe, the redness extends to adjacent parts, secretion does not accumulate in the follicles, and pus may be eventually detected. Sometimes the two occur together.

Treatment.—In the early stages, ice often relieves the pain; it should be sucked as well as applied to the throat externally. Salicylate of sodium in 10 or 15 grain doses every three or four hours may be given internally, and guaiacum lozenges (3 grains in each) may be sucked every two hours, to diminish the acute symptoms. If suppuration has commenced, hot fomentations and poultices probably hasten it. When pus is detected, an incision should be made into the tonsil with a bistoury, covered up to the last half-inch with plaster, so as to protect the other parts of the mouth. The patient is generally confined to bed, and can take nothing but fluid food. This should consist of milk, beef-tea, and strong broths; and stimulants are usually required. In later stages, quinine and iron may be given in full doses.

FOLLICULAR TONSILLITIS.

Ætiology.—This form of tonsillitis also recurs frequently in the same people, and arises from cold and from impure air.

Symptoms.—The tonsil is red and swollen, and presents several yellow prominent spots, which are follicles distended with secretion; and the surface is covered with more or less mucus. The swelling can be felt externally behind the angle of the jaw. In severer forms the secretion of the follicles is more abundant, and they are distended with large bright white plugs, which may present a close resemblance to the white material of diphtheria. Both tonsils are frequently affected. There is moderate constitutional disturbance, furred tongue, slight pyrexia, feeling of malaise, and the same local discomfort as in other forms of tonsillitis. Recovery always takes place.

Diagnosis.—*Diphtheria* is closely simulated when the plugs of secretion are large and white. Generally, their obvious formation within the follicle of the tonsil, or the existence of several on each side, serves to distinguish them. A single white patch of some extent, apparently only on the surface, is in favour of diphtheria, and an extension to the soft palate is conclusive. The bacteriological test of cultivation should be employed in doubtful cases.

Treatment should be nourishing and stimulating; quinine and iron, and port wine in weakly individuals. The tonsils may be painted with astringent or antiseptic solutions, such as glycerine of tannic acid, the liquor sodæ chlorinatæ, solutions of alum, or boric acid. Lozenges of chlorate of potash or rhatany may also be sucked.

CHRONIC ENLARGEMENT OF THE TONSILS.

Ætiology.—This is of common occurrence in children, without any apparent cause; some, it is true, are weakly in other ways, others maintain good health. Sometimes it can be traced to previous attacks of sore throat; on the other hand, those who have chronic enlargement of the tonsils are liable to temporary acute attacks. It often subsides as the patient approaches middle age, if not earlier.

Symptoms.—The tonsils are large, pale pink, lobulated on the surface, and firm in consistence. When only of moderate size, they may cause no symptoms. In other cases, the tonsils obstruct the passage of air from the nose through the pharynx, and the breathing is at all times somewhat snoring. From the same cause, the child breathes with the mouth open, and the nasal passages being little used, the anterior nares are small, and the alæ compressed. A more remote effect is the production of pigeon-breast, from deficient expansion of the anterior part and bases of the lungs when the ribs are yet soft and yielding. Swallowing is laborious and clumsy, and speech is suggestive of something being in the mouth. Hearing is also deficient, from catarrh of the Eustachian tube; and taste and smell are said to be affected. The same symptoms may, however, be due to adenoid growths in the naso-pharynx (hypertrophy of the gland tissue in the upper part of the pharynx—the pharyngeal or Luschka's tonsil), which frequently co-exist with enlarged tonsils. Cough, nasal catarrh, restlessness, and headache, are other symptoms observed in such cases.

Pathology.—The change in the tonsil is one of simple hypertrophy of all the component tissues.

Treatment.—The general health should be maintained by cod-liver oil, iron, and other tonics, including sea air. Local applications are of little service; but if the discomfort is considerable, the tonsils may be removed by the bistoury or tonsil-guillotine, after the local application of solution of cocaine. Adenoid vegetations are also removed by suitable forceps, or by the finger nail.

CHRONIC PHARYNGITIS.

Ætiology.—Chronic inflammation of the pharynx may arise from repeated acute attacks, but more frequently results from certain injurious influences, such as the abuse of alcohol, excessive smoking, and the continual use of the voice, as, for instance, in clergymen, singers, and hawkers. The condition is constantly associated with a similar change in the soft palate, tonsils, or posterior part of the nose.

Symptoms.—The mucous membrane may be reddened, with dilated veins; in some cases there are numerous small grey elevations scattered over the pharynx (*granular pharyngitis*); in others small abrasions or ulcerations occur. The grey projections in granular pharyngitis are the enlarged follicles or mucous glands. In some cases the mucous membrane is covered with increased secretion, and the patient is constantly hawking and spitting; in others the surface is dry, and a certain amount of discomfort and difficulty in swallowing, with pricking pain and desire to cough, is the result.

Granular pharyngitis is often spoken of as a distinct affection. It may spread beyond the fauces proper to the top of the pharynx and to the larynx; the mucous membrane is in most cases dry, but sometimes the follicles are covered with viscid mucus. It may cause little or no discomfort; but there may be stiffness and dryness of the throat, constant desire to hawk and spit, and distress and difficulty in swallowing. The effort to talk is also painful, and the patient may be obliged to stop to clear the throat. This condition of things is not uncommon in clergymen, public speakers, and others of like vocation, and has consequently been called "Clergymen's sore throat." The symptoms are aggravated by exposure to cold, and an inherited predisposition has been observed by some writers.

Treatment.—Local treatment is necessary in granular pharyngitis. Gargles are of little use, as they do not reach beyond the soft palate; but inhalations of alum or tannin may be employed, or the throat may be painted with astringent solutions, such as nitrate of silver (40 grains to the ounce), solution of perchloride of iron (ʒj to ℥j), or tannin (ʒj to ℥j), or iodised glycerine (iodine, 6 gr., pot. iod., 20 gr., ol. menth. pip., 5 min., glyc. to an ounce). If these fail, the granulations must be destroyed; and this is best done by the galvanic cautery or Paquelin's thermocautery, each nodule being successively touched. This may of course require several sittings; the resulting inflammation is checked by sucking ice for some hours afterwards. Anæmia, dyspepsia, and gouty tendencies must be met by suitable treatment.

RETROPHARYNGEAL ABSCESS.

This, though chiefly a surgical complaint, requires short notice here, since it is apt to complicate the diagnosis of some throat complaints, especially laryngeal obstruction. It arises often from caries of the spine, or inflammation of the post-pharyngeal glands; and it forms a swelling in the back of the pharynx, which may press upon the larynx so as to cause dyspnœa and asphyxia. Thus it may be mistaken for croup or laryngeal diphtheria, but the cough and voice are not husky and hoarse as in the latter, but rather "gurgling." In a doubtful case the finger should be passed to the back of the throat, when a fluctuating swelling will be felt. It should be opened by the surgeon.

DISEASES OF THE ŒSOPHAGUS.

ŒSOPHAGITIS.

The œsophagus is much less liable than other parts of the alimentary canal to the various forms of inflammation. It may be injured by chemical substances or hot fluids, or inflammation may extend to it from neighbouring parts. *Chronic* inflammation results from the pressure of tumours, and from valvular disease of the heart. It produces thickening and opacity of the epithelium, or actual warty growths, or in some cases dilatation of the veins and desquamation of the epithelium.

OBSTRUCTION OF THE ŒSOPHAGUS.

This is the most important pathological condition of this part of the alimentary tube. The causes are, impaction of foreign bodies, such as false teeth; compression from outside by mediastinal growths and thoracic aneurysms; the growth of cancerous or other tumours in the walls of the tube itself; constriction by the contraction of ulcers following injury by corrosive poisons; and functional spasm of the muscular walls. The last three conditions will be separately considered.

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in mind, and a careful examination for the symptoms of that disease should be made before the bougie is used.

Auscultation of the œsophagus may sometimes give assistance. It is effected by listening successively over each of the dorsal spines, while the patient swallows a mouthful of water, previously taken into his mouth. A peculiar gurgling sound is heard down to the point of obstruction, but not below.

Prognosis.—This is absolutely bad. Even if the obstruction is overcome, the malignant growth must be fatal by its further extension within a short time. The duration is generally from six to twelve months.

Treatment.—This is entirely mechanical. While a bougie, even of small size, can be still passed, some time may be gained by its use every two or three days; or the tube may be kept in permanently, and the patient fed thereby, a method that has been employed by Krishaber and Symonds. The latter uses a short tube, with a funnel-shaped upper end just projecting above the top of the stricture, and secured by a string which hangs from the patient's mouth. If these measures are inapplicable, the patient may be fed *per rectum*, or the stomach may be opened by the operation of *gastrostomy*.

Cicatricial Stricture.

In this again dysphagia is the main symptom; but it differs from cancer in this, that it may not advance beyond a certain point, and that it does not lead to any secondary effects, except dilatation of the tube above it. In consequence of this dilatation food often accumulates above the stricture, and is regurgitated after a time.

The **Diagnosis** is generally determined by the history, and the absence of other symptoms. Cancer would be excluded if the patient were young.

Treatment offers a fair chance of success if the sound can be passed through the stricture into the stomach. It should be used regularly once or twice daily, and attempts should be made to pass larger and larger instruments. Liquid food may be required always. In unfavourable cases gastrostomy may be advisable.

Spasmodic Stricture.

This occurs in nervous and hysterical young females, or even in males. There is difficulty in swallowing, accompanied by a painful sense of constriction in the throat and chest. The bougie may not pass at once, but steady pressure soon overcomes the difficulty. The neurotic condition of the patient must be treated.

DILATATION.

Dilatation of the œsophagus will follow any long-standing stenosis; the enlargement is spindle-shaped (or *diffuse*), but often largest at the lower end. A few cases have been recorded of diffuse dilatation without any obstruction. Dysphagia and regurgitation are the main symptoms in both groups of cases; in the latter it is probably due to muscular paralysis, and feeding by a stomach tube is the obvious treatment.

DIVERTICULA.

These are pouches in the walls of the œsophagus; they have been divided into (1) pressure diverticula, and (2) traction diverticula.

(1) *Pressure diverticula* arise from the impaction of foreign bodies, or from other local injury. As a consequence, apparently, the muscular coat is weakened, and the mucous and submucous coats are bulged out between the muscular fibres, which do not share in the coverings of the diverticulum. When once this has taken place food accumulates in the sac, which gradually enlarges, so that it may attain a diameter of three or four inches. It is usually hemispherical in shape. These diverticula are most common at the back of the œsophagus in its upper part.

The **Symptoms** are dysphagia, regurgitation of food, and foul breath from the decomposition of food in the sac. So much food may accumulate as to completely obstruct the œsophagus. The presence of such a sac is sometimes detected by the bougie being at one time obstructed, at another passing easily into the stomach.

Treatment must aim at supporting the patient by food introduced through an œsophageal tube.

(2) *Traction diverticula* are caused by adhesion of the œsophagus to surrounding parts, whereby the coats are pulled out in a funnel-shaped manner. They have occurred in children as a result of suppuration of the bronchial glands. They may give an opportunity for the impaction of foreign bodies, but otherwise have no clinical symptoms.

DISEASES OF THE STOMACH.

EXAMINATION OF THE STOMACH.

In the healthy individual the stomach lies almost entirely within the bony thorax, only the pyloric extremity being exposed in the abdomen, and that is partly overlaid by the left lobe of the liver. The pylorus itself lies three-quarters of an inch to the right of the middle line. The organ is recognised by the full tympanitic note which is yielded on percussion of the epigastric region, and the lower part of the left thorax in front. This area is limited above by the præcordial dulness, posteriorly by the splenic dulness and pulmonary resonance, and below by the resonance of the colon and small intestines; but the loudness and extent of resonance vary with the distension of the stomach, and sometimes an obvious gastric note can be elicited over the base of the left chest behind. In health, the greater curvature should not come below the level of the umbilicus.

In disease, the resonant area of the stomach may be abnormally extended by dilatation, or it may be limited by the growth of a tumour near the pylorus, or by the enlargement of either liver or spleen so as to cover a larger portion of its area; it may be displaced downwards by tumours or liquid in the chest, and upwards by ascites, or by shrinking of the left lung.

Besides mere physical examinations, some information may be obtained by the examination of vomited matters, and of fluids artificially withdrawn from the stomach during the process of digestion. To this latter method much attention has been paid of late, both under normal and pathological conditions, and it has thus been sought to ascertain the share which a deficiency of the acids, of the pepsin, or of the motor powers of the stomach may have in different forms of disease, especially the chronic disorders of digestion. The application of these tests in ordinary clinical work is troublesome, and the results are not always trustworthy, but in occasional cases it may be desirable to employ them.

The principle mainly followed has been to wash out the stomach (*see* Dilatation), then to introduce a small quantity of food—for instance, about 1½ ounce of bread and 10 ounces of water—and to withdraw the contents after an interval of half an hour, one hour, or more. The fluid is then analysed with reference to its total acidity by titration with an alkali, and to the respective amounts of hydrochloric acid, lactic acid, and of peptones it

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GASTRIC IN

Indigestion or dyspepsia—that
by the gastric secretions, or pain
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affections of the stomach; and
nearly all kinds of indigestion as
gastritis. But more
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ACUTE INDIGESTION.

This is a purely functional or mechanical disturbance, from the ingestion of food in too great quantity, or of specially irritating quality. Any one in perfect health may be tempted to take a larger quantity of food than his stomach can bear; or, with an ordinary quantity of food, some ingredient, such as ice or coffee, or alcoholic drink in excess, may be taken, which retards the process of digestion, and the whole quantity ingested remains for some hours in the stomach. Or the unexpected failure to digest may be due to preceding general exhaustion, in which the stomach shares; for instance, after excessive exercise in walking or climbing during several hours without refreshment, the stomach may fail entirely to digest even a moderate meal put into it.

Symptoms.—Either at once, or within a few hours of the meal, there is a sense of distension and discomfort, or actual pain, in the gastric region; if the offending meal has been a late dinner the patient may go to sleep with little trouble, but wakes after a few hours with gastric distress, a dry tongue, and perhaps headache, and may lie wakeful for some hours. Sometimes a quick fluttering is felt from time to time at the præcordial region, or the heart actually intermits a few beats. In the morning there is little inclination for food, the tongue is dry and furred, and the skin clammy; but in the course of a few hours the symptoms subside. In other cases the illness is more quickly terminated by the occurrence of vomiting, and the stomach is generally emptied of the whole of its contents, which are only partially, if at all, digested, and are mixed with gastric mucus. The pain is often at once relieved, and the patient falls to sleep. At other times the vomiting may be repeated, and bile, which has been regurgitated from the duodenum, may be discharged with the later efforts. Sometimes in the course of the next twelve hours the bowels are actively moved, from the passage into them of undigested or irritating materials.

Treatment.—Where the pain is severe, and the cause is obvious, immediate relief may be obtained from an emetic such as ipecacuanha or sulphate of zinc; if it fails to act, apomorphine by subcutaneous injection may be desirable. In milder cases it is sufficient to quench thirst with a very little ice, and to abstain from introducing anything further into the stomach until the distressing symptoms have subsided.

CHRONIC INDIGESTION.

Ætiology.—The process of digestion is impaired if the *food* be excessive in quantity, or if it be improperly masticated from defective teeth, or from too great haste to swallow. Some foods are less soluble in the gastric juice, such as coarse-fibred meats, pulpy fruits, or stringy vegetables where cellulose is in excess. Pork, veal, game, and others mentioned under gastritis are among these.

On the part of the *stomach* we may note that all the organic lesions are causes of dyspepsia; but that apart from these indigestion arises from excess or deficiency of gastric juice, from deficiency of hydrochloric acid, or of pepsin, or from excess of mucus. Weakness or degeneration of the walls of the stomach, and compression or displacement by adjacent viscera, the descent of the diaphragm, a pregnant uterus or other abdominal tumour impair its motility, and thus prevent a proper admixture of the contents. Constipation has been thought to have a reflex influence in the same direction.

General illnesses and other causes of low vitality, such as anæmia, phthisis, and the infectious fevers, affect both secretion and motility.

Symptoms.—These are very variable in different cases.

Local sensations.—Indigestion is shown frequently by pain in the epigastric region, which comes on after taking food, and lasts a certain time, gradually subsiding. It may be strictly localised, or radiate to the left, or extend to the præcordial region (*cardialgia, heartburn*), or be felt between the shoulders, going "through to the back." Sometimes, as digestion slowly proceeds, the pain extends to the umbilicus or lower abdomen. In other cases pain begins when the stomach is empty, and is relieved by ingestion of food. Instead of pain there may be only a sense of discomfort, tightness, or fulness.

Gastralgia and *Gastrodynia* signify simply pain in the stomach, and are commonly used to designate gastric pain, from whatever cause arising. Some writers reserve these terms for severe forms of pain, which are spasmodic in character, may be independent both of organic disease and of the digestive process, and are possibly neuralgic in origin. Such pains occur often in females, and sometimes in association with gout. Others, again, would arbitrarily define gastralgia as pain in an empty stomach, relieved by food; and gastrodynia as paroxysmal pain independent of food.

Flatulence.—The excessive formation of flatus is a common result of dyspepsia. The gastric region is distended, much discomfort and pain are caused, and waistbands may have to be loosened. Some relief is afforded by eructations, and after a time by the passage of the wind *per anum*. *Borborygmi*, or noises produced by wind passing along the intestine, occur at the same time. Flatulence and borborygmi also occur occasionally on an empty stomach. In most cases, no doubt, flatus is due to bacterial fermentation of the contents of the stomach, as a result of a diminished secretion of hydrochloric acid, which normally prevents the action of bacteria. In other cases the air has been swallowed, or it enters the stomach from the intestines, or it is exhaled from the blood-vessels of the stomach.

Nausea is an occasional symptom of dyspepsia, and *vomiting* less frequent, except in drunkard's dyspepsia (*see* Chronic Gastritis). The vomited matter is either the ingested food, or merely mucus. With repeated emesis bile may be rejected, and a few streaks of blood; but a large amount of blood is quite rare. *Pyrosis*, or *water-brash*, is the name given to a condition in which a quantity of neutral or alkaline liquid is brought up into the mouth; this is commonly believed to consist chiefly of saliva. A burning pain in the epigastrium is present at the same time.

General Symptoms.—The tongue is variable; it is often furred, and is large, pale, and flabby, or red, narrow, and pointed. The fur may be thin and white, or thick and yellow or brown. Constipation is frequent; but may be interrupted by occasional diarrhœa. Some cases are characterised by the rapid passage of undigested food through the intestines (*lienteria, lienteric diarrhœa*). The urine will vary with the amount of water ingested, and with the presence or absence of vomiting; and deposits, sometimes of urates, at others of phosphates, are likely to occur. Malaise, indisposition for exertion, slight anæmia, or sallowness, some loss of nutrition, and in chronic cases a settled expression upon the face of discomfort or anxiety, are not uncommon. But in other instances there is no general indication whatever of the gastric fault.

Varieties.—The several classifications of dyspepsia are not altogether satisfactory. Some refer to the cause, and thus we hear of *atonic* dyspepsia, *gouty* dyspepsia, *renal* dyspepsia, and *irritative* or inflammatory dyspepsia, which is only another name for gastritis, or gastric catarrh. Or the indigestion is named after the results—such as *flatulent* dyspepsia, or *acid* dyspepsia; but flatulence and acid eructations may occur in other conditions than mere indigestion. A classification as to cause is of value, as indicating in what direction to look for treatment, but the form of indigestion may be practically the same in many instances.

Atonic dyspepsia arises in various conditions of depression of vital power, is common in anæmic persons, and appears to depend on a want of functional power of the stomach, in regard both to secretion and to muscular contraction. The tongue is broad, flabby, and indented by the teeth, there is little pain after food, except as a direct result of flatulent distension, and the appetite is bad, but there is no thirst. In some of these cases there is a deficiency of free hydrochloric acid.

Flatulent dyspepsia is a term which may be given where the

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Treatment.—Indigestion requ
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veal, game, shell-fish, pastry, carrots, turnips, and parsnips. Boiled or roast mutton, fish, or chicken may be allowed; but in severer cases the diet should be confined to milk or peptonised milk, or milk and farinaceous articles. If acid fermentation is a marked feature, farinaceous foods should be limited, and milk and fish given. The liquid irritants, alcohol, tea and coffee should also be eschewed. Sometimes repeated experiment alone will show what foods the patient can tolerate; but constipation should be relieved; and the patient should avoid all mental worries and overstrain, or business anxieties. Where atony, debility, or nervous prostration is a prominent feature, complete rest is of great value; in other cases sufficient but not exhausting exercise should be taken. Various drugs are useful, especially the alkaline carbonates given before meals; bismuth; the mineral acids; the bitter tonics, calumba, gentian, and nux vomica; and carminatives, such as sp. ammon. arom., cardamoms, and ginger. Slight cases of oppression after meals are often benefited by dilute hydrochloric acid, which supplies the defective secretion in the stomach, and nux vomica; in flatulence and constipation, rhubarb, soda, and calumba, and rhubarb and magnesia are of value. Flatulence alone may be lessened by bismuth or sodium sulpho-carbolate before meals, by ginger, peppermint, cardamoms, and other carminatives, or by creosote and charcoal; and acid dyspepsia by alkalies, especially sodium bicarbonate, given an hour or two after meals, or bismuth lozenges. As improvement takes place, nux vomica, or strychnia with quinine is useful in giving tone to the stomach and the system generally. In gastralgia or gastrodynia, and all cases with intense pain, opium or morphia may be administered in small doses, and belladonna liniment and hot fomentations should be applied to the stomach; or, in severe cases, a small blister.

NEUROSES OF THE STOMACH.

Many purely nervous symptoms have already been mentioned under indigestion, and the connection is obvious. Nervous conditions have intimately to do both with the secretion of gastric fluids and with the motor power of the stomach, on which perfect gastric digestion so much depends. *Gastralgia* and *gastrodynia* are sometimes no doubt independent of digestive defects, and rather neuralgic in their origin, but they are often difficult to discriminate from the pains of gastric ulcer, and genuine sufferers from the latter have often been wrongly regarded as hysterical. The *gastric crisis* of locomotor ataxy is another form of pain which, with its attendant vomiting, is a gastric neurosis. *Vomiting* is often hysterical or neurotic, and entirely independent of ulcer, cancer, or gastritis. It may occur directly food enters the stomach; it may be accompanied or preceded by pain. The known characteristics of the patient or the maintenance of health in spite of the symptoms may save one from errors. Children are not unfrequently the subjects of this difficulty. The vomiting of brain disease is another illustration. *Bulimia* (excessive appetite) and *anorexia* (deficient appetite) probably have their seat in the stomach. Deficient appetite is common in hysterical females, and *anorexia nervosa* is an extreme form, already mentioned (*see* p. 356). *Flatulence* in some of its forms develops with such rapidity that it is not easy to explain it on chemical theories; but though its relation to other neurotic conditions is a frequent one, the method of its production is not so obvious.

GASTRITIS.

ACUTE GASTRITIS.

Ætiology.—Acute inflammation of the stomach, or acute gastric catarrh, may be set up by various forms of irritants. The most intense form of gastritis occurs in poisoning by the strong mineral acids, or other corrosives, and will not be here considered. The more common cases arise in consequence of the use of indigestible food, such as lobster, crab, or shell-fish, or of unripe fruit, or of flesh, fish, fruit, vegetables, or other food, which is in a state of commencing decomposition. It is thus frequent in hot weather, and may be further contributed to by the ingestion of large quantities of water. Infants frequently suffer in this way from unsuitable food, which is in different cases the mother's milk, or cow's milk insufficiently diluted, or one or more of the various starch-foods and biscuit-foods in use.

Symptoms.—There is a feeling of weight, or oppression, at the epigastrium, and in many cases actual pain, which is increased by pressure, or by the ingestion of food. With this there are nausea and retching; or vomiting is at once produced by the introduction of anything into the stomach. The vomited matters at first consist of particles of food, afterwards they are watery or mucous, or stained with bile. They contain a diminished quantity of hydrochloric acid. The patient is dull and heavy, with some headache, generally constipation, loss of appetite, thirst, an unpleasant taste in the mouth, a thickly furred tongue, and offensive breath. There is sometimes decided febrile reaction, and the pulse is soft and quick. Examination of the abdomen shows that the epigastric region is hard and tense, and sensitive to pressure.

Lebert describes a mild form of gastric catarrh, in which there is no fever, almost no pain, and rarely vomiting, but the other features are much the same as in the severer forms.

In infants, the complaint is generally associated with diarrhœa. The little patient is constantly fretting or whining from pain, the legs are drawn up to relieve it, the abdomen is tense and tender, food is either refused, or, if taken ravenously to quench thirst, is as rapidly rejected, emaciation soon occurs, and death may be the result. Except in the case of infants, the disease generally subsides in the course of a week or two, but repeated attacks may lead to a chronic condition.

Morbid Anatomy.—In the majority of cases nothing can be known of the condition of the mucous membrane of the stomach, since recovery takes place; and the changes which have been found in the stomach after death from the infectious diseases must not too readily be assumed as identical with those occurring in ordinary cases, since there is an absence of the symptoms characteristic of these last. But in the well-known case of Alexis St. Martin it was shown that changes quickly followed irritation of the mucous membrane. Red pimples appeared, which were sometimes filled with purulent matter, or there were red patches, or aphthous crusts, or abrasions. The gastric juice was secreted in less quantity, and mucus was poured out freely. Slight hæmorrhage also occurred sometimes. Ziegler states that in gastritis the mucous membrane is dark red and swollen, beset with small hæmorrhages, and covered with a film of mucus, mucoid epithelium, and extravasated leucocytes. The cylindrical epithelial cells of the gland ducts are in an extreme stage of mucoid change, and many desquamate; and the epithelial cells of the peptic glands are detached, and seem more granular than usual. The vessels of the interglandular tissue are distended; and the subglandular tissue and even the submucous layer are infiltrated. Abrasion and ulceration may also occur, often perhaps at the seats of previous hæmorrhages; and as later results, induration and atrophy of the mucous membrane supervene.

Diagnosis.—This is generally simple. It has, however, been pointed out that acute gastric catarrh may be confounded with the early stages of *enteric fever*, and with *peritonitis*. The former difficulty may not be cleared up until the appearance of rose spots and a typical diarrhœa; in peritonitis we should expect more generally diffused pain, a more rapid onset, greater distension and early collapse.

Treatment.—It is of the first importance to give complete rest to the stomach. In severe cases food should be stopped altogether for a time, and as little as possible of any kind should be introduced into the stomach. If the symptoms do not abate in twenty-four hours, nutrient enemata may be employed. In milder cases, very small quantities of milk and soda-water may

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The **Treatment** must be mainly symptomatic, unless the presence of a definite swelling should give a chance for operative interference.

CHRONIC GASTRITIS.

Ætiology.—Chronic gastritis may be the result of an acute attack, but more often it arises from the continued ingestion of irritating or indigestible food, such as pork, veal, pastry, fruit, or tea and coffee in excess; and it is a constant result of undue indulgence in alcoholic liquors. Local conditions of the stomach may also cause it, such as the venous congestion which results from diseases of the liver and heart, and the irritation of malignant disease or chronic ulcer. Almost all conditions by which the processes of digestion and the preparation of the food for digestion are interfered with may be causes of gastritis, though they often do no more than induce the functional disturbance known as indigestion or dyspepsia. Such are defective mastication, bolting the food, irregularity in taking meals, mental anxiety, overwork, and other debilitating influences, such as prolonged illness, fever, phthisis, or Bright's disease.

Morbid Anatomy.—In very chronic cases, the wall of the stomach is generally thickened, and presents various degrees of vascularity, not always very marked. Numerous dark or slate-coloured patches of pigmentation give evidence of former congestion or hæmorrhage; and occasionally small ulcers, *hæmorrhagic erosions*, are scattered over the surface. Sometimes the mucous membrane is atrophied entirely, at others there is fibrous overgrowth of the interglandular, submucous, and intermuscular connective tissue, while the glands disappear or become cystic, and the muscular fibres perhaps waste. The process of thickening sometimes produces, especially in the pyloric region, a rough and wrinkled surface, commonly described as *mammillated*.

Symptoms.—There is generally some tenderness on pressure in the epigastric region, but pain is not often severe. It may be aggravated by food, and is felt in the epigastric region, and perhaps in the back between the shoulders, or there is a burning sensation internally. Nausea is more frequent, and there is sometimes vomiting. Vomiting is the most prominent feature of the gastritis of drunkards, and occurs in the morning immediately the patient rises from bed. The vomited matters mostly contain a good deal of mucus, but rarely blood; sometimes they are acid, and contain butyric, lactic, and acetic acids from fermentation in the stomach, but the hydrochloric acid is deficient. Flatulent distension of the epigastric region, and eructation of gas may also be present. The associated conditions are decided thirst, capricious and often deficient appetite, offensive breath and unpleasant taste; a furred tongue, red at the tip and edges,

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Treatment.—It is of th
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charcoal. Severe pain may require locally hot fomentations, or a small blister, or opium or morphia internally.

ULCER OF THE STOMACH.

Ulceration of the stomach occurs in several forms. In that which has the greatest clinical importance, and is generally meant by the term "gastric ulcer," the lesion is mostly single, has a very chronic course, reaches a large size, gives rise to severe symptoms, including pain, vomiting, and hæmorrhage, and is often fatal by exhaustion, perforation, or otherwise. Slighter forms of ulceration occur in the course of chronic gastritis, and in consequence of hæmorrhage into the mucous membrane, whereby the gastric juice is enabled to act upon and dissolve its superficial layers. These are described as *hæmorrhagic erosions*, and arise in conditions of congestion of the mucous membrane, as in cardiac disease, in emphysema of the lungs, or in portal obstruction.

Ætiology.—Chronic ulcer of the stomach, though seen at all ages, except in childhood, is very much more frequent between the ages of twenty and forty. Clinically, it is much more frequent in women than in men, in the proportion of three to one; but it seems to be fatal to the sexes in about equal proportion. It is seen more often in the poorer classes, and largely amongst female servants; it is also associated often with chlorosis and anæmia, but beyond this its predisposing conditions are not very obvious.

Morbid Anatomy.—The chronic ulcer, as it is seen after death, is round or oval in shape, measuring from one-third to four or five inches in diameter; though more often single, there may be two or more, and two may coalesce so as to form an irregular-shaped ulcer of large size. In the early stage the ulcer has thin edges and is rather shallow; in later stages it extends deeply into the muscular coat, or even through this so as to expose the serous coat; and it is often more or less conical or funnel-shaped, the destruction of the mucous membrane being more extensive, that of the sub-mucous tissue less so, and that of the muscular coat least. The edges are often thickened and raised above the surface, from infiltration with inflammatory material; and the thickening extends some little way into the surrounding mucous membrane.

The position of the ulcer is of importance; in more than half of the cases it is in the neighbourhood of the pylorus, or on the lesser curvature; whereas the cardiac orifice, the cardiac extremity, and the greater curvature are much less often the seat of it. It is more frequent on the posterior than on the anterior surface.

When the ulceration reaches the peritoneum this may ruptur so that perforation takes place, the contents of the stomac escaping into the peritoneal cavity and setting up intense genera peritonitis, or a more localised abscess containing air (*subphreni abscess*). More often the inflammatory process, extending to th serous surface, causes the stomach to adhere to one of th adjacent parts before perforation can occur. This is most frequently the pancreas or the left lobe of the liver, but adhesio also takes place occasionally to the diaphragm, spleen, colon anterior abdominal wall, and even the supra-renal capsule. Th ulcerative process then extends into the newly attached organ and large cavities may be formed in the liver and pancreas Thus also the diaphragm may be perforated, with subsequent pleurisy and pneumonia; and the colon may be opened, or th abdominal wall invaded with the formation of gastro-colic fistula in the one case, and gastro-cutaneous fistula in the other. Rarely an old ulcer causes so much adhesion and matting together of th parts, that a cancerous tumour is closely simulated. Hæmorrhag is a common accident, mostly from gastric vessels in the wall o the ulcer, but sometimes from the splenic artery after adhesion to, and ulceration of, the pancreas. Occasionally the advancing ulce sets up a localised suppuration in the peritoneal cavity.

But all ulcers are not so destructive; many recover completely. If they have been small, they leave no scar; if of large size, the scars are thick and puckered, and may themselves give rise to considerable trouble. Thus, at and near the pylorus they may by their contraction cause stenosis, and consequent dilatation of the stomach; occasionally the stomach is contracted across its middle (hour-glass contraction). Lastly, it must be mentioned that old ulcers sometimes become cancerous.

Pathology.—No satisfactory explanation has yet been given of the occurrence of gastric ulcer. The most commonly accepted view has been that a small vessel has been obstructed by embolism or thrombosis, and that the corresponding part of the mucous membrane, deprived of its alkaline blood, is dissolved by the acid gastric juice. S. Martin ascribes it to bacterial necrosis, especially as the ulcer occurs in the pyloric region, where the antiseptic action of hydrochloric acid is likely to be less powerful. Injury, chemical agents, and scalding food, have been suggested as causes, but will not explain more than a few cases.

Symptoms.—In a small number of instances there are no symptoms at all, and the affection is quite latent, until the patient is suddenly taken with vomiting of blood, or *hæmatemesis* In many others there are symptoms differing but little from those of gastric catarrh, or other form of dyspepsia, consisting of pain after food, distension, and occasional vomiting. But the typical cases are distinguished by the characters of the pain and th presence of blood in the vomit. The *pain* is situated in the epigastrium, just below the ensiform cartilage, sometimes nearer the umbilicus, or to the right or left of the middle line—the right more often than the left. It is generally brought on by the ingestion of food, appearing from half an hour to two hours after a meal; it may continue intense until vomiting takes place, by which it is generally relieved, or it subsides as the food leaves the stomach. In character it is pressing, boring, tearing, or burning, and more severe than in any other gastric disorder. Sometimes there is pain in the back, between the eighth dorsal and the second lumbar vertebræ. There is often tenderness and hyperæsthia over the epigastrium.

The vomited matters may be only the gastric contents, but in the majority of cases hæmorrhage takes place. In some instances the blood is exuded in small quantities, and mixes with the contents of the stomach, so that the hæmoglobin is converted into hæmatin by the acid gastric juice, and the vomited matter has the turbid, blackish-brown appearance, which has been compared to *coffee-grounds*. In others the blood may be extravasated in large quantity at once, flowing freely from a large artery, so that it is promptly vomited, unmixed with gastric contents, and retaining its arterial brightness. The patient, who may have never brought up blood before, feels faint, has a sense of oppression in the epigastrium, and in a few minutes vomits the blood, which may amount to one, two, or three pints.

Some of the blood discharged into the stomach finds its way into the intestine, and is ultimately passed *per rectum* in the form of black, treacly, or tarry motions, constituting *melæna;* these may appear some hours after the hæmatemesis has ceased. The vomiting of pure blood may continue so as to be fatal; more often it ceases entirely, and may not be repeated for months or years, if at all. High degrees of anæmia and weakness result from the loss of blood. Occasionally, when hæmorrhage occurs no blood is vomited, but the whole passes *per rectum.*

The continued pain, the defective assimilation of food from vomiting, and the loss of blood, naturally impair the general condition of the patient earlier or later; but there is no fever, the tongue is clean, unless there is much gastric catarrh, and the appetite is often very good. Constipation, however, is frequent. Examination of the abdomen generally reveals nothing; there may be some hardness, or tenseness of the abdominal walls. Only in the case of old ulcers with much thickening, or adhesion to other organs, can anything like a tumour be felt; and if pyloric stenosis results, the dilated stomach may be recognised. The hydrochloric acid of the stomach contents is often in excess; but sometimes it is diminished. After hæmatemesis there is a characteristic pallor, with the usual hæmic murmurs over the præcordia (*see* Anæmia).

The symptoms of gastric ulcer are undoubtedly very amenable to treatment, and *post-mortem* results as well as clinical records show that recovery often takes place; this, however, generally requires the continuance of treatment, and especially the judicious avoidance of harmful ingesta, for a very long period. Many patients, after months of freedom from symptoms, are again severely affected. In the more serious cases pains and vomiting are constant, and much blood is lost in the coffee-ground matters ejected. Ultimately death takes place by exhaustion. Large hæmorrhages, though frequent, are comparatively seldom the direct cause of death. Perforative peritonitis generally causes sudden acute pain, followed by severe collapse, and rigidity of the abdominal wall; or in some cases distension with air which has escaped into the peritoneal cavity. Perforation of the diaphragm leads to fatal empyema or pneumonia; but gastro-cutaneous fistula is not in itself dangerous, and may persist for years.

Diagnosis.—The most important element in the diagnosis is hæmatemesis. Pain, vomiting, flatulence, &c., may be quite inconclusive, until the discharge of a large quantity of blood gives evidence of the ulcerating process. Hæmatemesis occurs also in cirrhosis of the liver, and in cancer of the stomach; *cirrhosis* may often be shown by the age of the patient, his or her known alcoholic habits, and the facial aspect. Drunkards also suffer from gastric disturbance, but mostly in the form of vomiting or retching in the morning, when the stomach is empty; nor is pain a prominent feature. The hæmorrhage of *cancer* is rather in small quantities often repeated, than in large quantities at one time. A tumour is generally present, and the cancer patient is rarely under forty years of age. Hæmatemesis must, of course, not be confounded with hæmoptysis (*see* pp. 459, 468), nor with vomiting of blood after epistaxis.

In the absence of hæmorrhage, the diagnosis is not always easy; sometimes ulcer can only be suspected. A severe localised pain of weeks' duration, aggravated by food, relieved by abstinence or judicious diet, accompanied by local tenderness, a clean tongue, absence of general disturbance other than can be explained by the pain and vomiting, a patient between seventeen and thirty-five, are grounds for the diagnosis and treatment of gastric ulcer. The diagnosis from cancer will be further considered in the next chapter.

Prognosis.—This must always be doubtful, since neither hæmorrhage nor perforation can be foreseen. The earlier a case comes under treatment the more likely it is to be favourable. A long duration of severe dyspeptic symptoms and frequent coffee-ground vomit are unfavourable. A profuse hæmorrhage indicates deep ulceration, but its very severity may compel treatment, which the patient otherwise would not have submitted to, and so in the end, perhaps, be of value. Dilatation of the stomach is, of course, unfavourable.

Treatment.--The most important indication is to give the stomach as much rest as possible; if the pain is severe and vomiting is frequent, food should be given for a few days entirely by the rectum. After a time, or from the beginning in the less severe cases, it may be given by the mouth. This should be at first nothing more than milk, an ounce or two every two or three hours; if this is not well borne it may be mixed with one-third of its quantity of lime-water, or of soda-water; or, better, it may be milk peptonised by the addition of the liquor pancreaticus of Benger. In any case, the quantity introduced at one time into the stomach must be small, and such as will not induce pain or vomiting. The food may afterwards be increased, so that the daily amount gradually rises from one pint to two or two and a half in the twenty-four hours. It is not generally desirable to exceed the latter quantity. The next step should be to thicken the milk with arrowroot, ground rice, or biscuit powder; and beef-tea or other meat solution may be added. Only when the symptoms have entirely subsided for some days, should more solid food be allowed, and this may begin with fish, chicken, or raw pounded meat. Vegetables and fruit must be avoided almost throughout.

How far drugs directly influence the healing of the ulcer is doubtful, but they may be required to meet particular symptoms. For pain, if unrelieved by diet, one may give opium in small doses of the extract or tincture, or the liquor morphinæ hydrochloratis in 10 or 15 minim doses. In severe cases the hypodermic injection may be used; but the opiate treatment must always be discontinued as soon as relief is obtained. Bismuth is also of value; 10 or 15 grains of the subnitrate suspended in mucilage should be given before meals, and the dose may be increased to 30 grains. It may be combined with morphia. Local applications to the epigastrium may be used, such as hot fomentations, mustard leaf, or even blisters in severe cases; sometimes ice-compresses give relief. Heartburn may be relieved by alkalies, especially bicarbonate of sodium; constipation by cold water enemata, or Carlsbad salts in the morning before breakfast; by compound liquorice powder, or pills containing rhubarb or aloes. Vomiting may be checked by morphia or bismuth and morphia, by effervescing medicines, or by a few drops of tincture of iodine in two drachms of water every hour.

If a profuse hæmorrhage occurs the patient must be kept at rest, and no food must be given by the stomach for some hours;

ice should be applied to the epigastrium; and ergotin may be injected in 1 or 2 grain doses every two or three hours. With continued discharge of blood other astringents should be used internally, such as tannin, acetate of lead, alum, or tincture of perchloride of iron in 5-minim doses every hour.

If a patient known to suffer from gastric ulcer is seized with the symptoms of perforation (p. 612) the abdomen should be opened as soon as possible—*i.e.*, within a few hours—the peritoneal cavity washed out, and the ulcer sutured.

CANCER OF THE STOMACH.

Ætiology.—Cancer of the stomach is rarely seen before the age of thirty, and the majority of cases occur between forty and sixty. Sex has no appreciable influence; and though there can be no doubt that cancer in general is to a certain extent hereditary, this is not a prominent feature of cancer of the stomach. Lebert gives seven per cent. as the proportion of cases that can be attributed to hereditary influence. It is equally frequent among the rich and the poor, and is not related to any particular occupation. It frequently happens in those who have been hitherto quite healthy, and is not determined by any previous disease of the stomach, with the exception of ulcer, a few cases of which have been found in association with cancer, or actually themselves becoming cancerous.

Pathological Anatomy.—Cancer affects all parts of the stomach, but in the majority of cases the pylorus is involved, and the disease extends thence to the adjacent parts of the organs; especially along the lesser curvature. If it affects the cardiac end the œsophagus is generally also invaded. Sometimes the wall of the stomach is uniformly infiltrated and thickened, and the organ, as a whole, is contracted to a small size. With few exceptions cancer of the stomach is in the form of spheroidal carcinoma, or cylindrical carcinoma; and the former is much more common. Either variety may be scirrhous from excess of fibrous tissue, or medullary from deficiency of it; and colloid degeneration may take place in either, but is more common in the spheroidal variety. The scirrhous change is the most common. Squamous epithelioma and sarcoma are comparatively rare.

The cancer commonly begins as an overgrowth of the epithelial cells of the glands of the mucous membrane; the growths project into the submucous tissue, proliferate further, and gradually involve all the coats. In cancer of the pylorus the whole wall of the stomach at this spot is thickened, and projects internally so as to narrow considerably the passage from the stomach into the duodenum; this may admit with difficulty the little finger or a large catheter. The projection terminates abruptly towards the duodenum, more gradually towards the stomach. The thickening mostly affects the submucous layer, but also the muscular coat; and the bands of muscular fibre are separated from one another by the new growth. Subsequently the subserous layer is involved, and deposits of cancer may occur in the adjacent peritoneal surface; in later stages it often ulcerates upon the inner surface. The adjacent mucous membrane may show nodular growths or villous processes.

Important changes occur in the stomach and adjacent parts as a result of cancer, which are in many ways similar to those following simple ulcer. Thus, the ulcerative process may erode vessels and lead to hæmorrhage; this is much less often profuse than it is in simple ulcer. Dilatation of the stomach is very frequent as a result of the narrowing or *stenosis* of the pylorus, which the growth of cancer necessitates; but in a certain number of cases the stomach is actually smaller, and this generally when the cancer affects the whole of the organ, as already shown. *Adhesion* of the stomach to other organs commonly takes place, as the growth reaches the peritoneal surface, and invasion of the organ with cancer may follow. The liver and pancreas are thus frequently attacked; occasionally the spleen or colon. In the last case a *gastro-colic fistula* may result. When the cancer is in front the abdominal wall may become adherent, or, in the absence of adhesion, perforation into the peritoneum may take place; but this is much less likely than in simple ulcer. More often subacute or chronic *peritonitis* takes place without perforation, either spreading from the original lesion, or following a general growth of cancer in the peritoneum.

Secondary deposits occur in various organs, in the peritoneum, as just stated, in the liver, pancreas, and especially in the mesenteric, retroperitoneal, and portal lymph-glands. The spleen is more rarely affected; and occasionally more distant organs, as the brain and lungs. After death from cancer, the heart is commonly in the condition known as *brown atrophy*.

Symptoms.—In the earliest stages there is nothing characteristic about the symptoms, which are chiefly those of dyspepsia. There is discomfort, fulness, weight, or pain after food, with acid eructations or flatulence. The pain may be at the epigastrium, or in the position of heartburn; small quantities of food may be regurgitated. After a time *vomiting* takes place, at first only at long intervals, then weekly, or two or three times a week. *Pain* then becomes a more prominent symptom, and though, like the other symptoms, at first related more or less closely to the ingestion of food, it soon becomes more constant, or arises independently of a meal.

It commonly radiates from the epigastric region, where it is, in later stages, most intense, and is occasionally felt between the shoulders, or in the lumbar region. It is often stabbing, and lancinating, but may be boring, burning, gnawing, or tearing. The vomited matters consist of food in different stages of digestion, mixed with more or less mucus, or streaks of blood. Often the blood mixed with the vomit has been altered by conversion of hæmoglobin into hæmatin, so as to give it the characteristic coffee-ground appearance. Abundant hæmorrhages are much less common than in ulcers; but if a large vessel, such as the splenic artery, is eroded, profuse and fatal bleeding will result.

In the majority of cases, a *tumour* is discovered at some time or other; but rarely in the first three or four months. The position of course varies with the part of the stomach affected. A pyloric tumour is commonly situated in the middle line, or a little to the right, midway between the ensiform appendix and the umbilicus, but it may be close to the umbilicus, or more to the right of the middle line; indeed, almost anywhere in the triangle formed by the right costal margin, the middle line, and a horizontal line running through the umbilicus. When the stomach is much dilated the tumour is even below the umbilicus. It varies in size from that of a walnut to that of a small orange, is generally very hard, sometimes globular, but often somewhat square, and mostly irregularly nodular. It is at first freely movable, and descends on inspiration, but in later stages it may contract adhesions and become more fixed. It frequently receives an impulse from the underlying aorta. On percussion it is dull, or imperfectly resonant; handling it causes pain, which may last for some time afterwards.

The condition of the abdomen varies. As a result of the small quantity of food that passes the pylorus, it is frequently empty, but presents in many cases the prominence in the upper or left portion, which is due to dilatation of the stomach. Dilatation is further recognised by the peristaltic wave of contraction, the varying resonance and dulness on percussion according to the amount of the contents, the splashing sounds heard on movement and the characters of the vomited matter (*see* p. 621).

In some cases the amount of fibroid change in the pylorus is so great that the lesion has been regarded as purely fibrous, and called *hypertrophic stenosis*. But the invasion of lymphatics with cancer shows their origin. However, the progress of these cases is slow, and the patients for years suffer only from the resulting dilatation.

Though the pylorus is very frequently the seat of gastric cancer, it must not be forgotten that it occurs in other situations, and that there will be some differences in the symptoms and physical signs in accordance with these. Thus, a tumour in the back of the stomach may grow to a large size without being felt; or it may be felt, but mistaken for a kidney. The signs yielded by a general infiltration of the gastric walls are very obscure. In all these cases dilatations are not present as they are in so many cases of pyloric cancer. The pain may be also rather in the left loin or back, than in the epigastrium.

Indications of severe constitutional disturbance set in comparatively early in cancer of the stomach. The appetite diminishes, the patient loses flesh, strength, and colour; and in advanced conditions *emaciation* and *anæmia* are extreme. The tongue is often clean, but in the last days may be dry and covered with sordes or deposits of thrush. The bowels are mostly constipated, especially if dilatation supervenes.

Various complications may arise towards the end. Ascites may follow the spread of cancer to the peritoneum, or the implication of the liver and portal vein. Cancer of the retroperitoneal glands causes œdema of the feet; or the same is brought about by thrombosis of the large veins.

Gastro-colic fistula, which is mostly the result of cancer spreading from the stomach to the colon, is marked either by *lienteria*, undigested contents of the stomach passing directly into the colon, and hence *per rectum;* or by fæcal vomiting, the contents of the colon passing into the stomach and hence being vomited. The former is more frequently the case when the pylorus is obstructed.

Death commonly takes place from exhaustion, as a result of continued pain, vomiting, and deficient assimilation of nutriment. Rarely a profuse hæmorrhage, or peritonitis, bronchitis, or pneumonia may terminate the scene.

Duration.—The illness commonly lasts from six months to two years; two-thirds of the cases last less than eighteen months, and a very small proportion more than two years.

Diagnosis.—Cancer is usually distinguished from the majority of the diseases of the stomach by the presence of a tumour. This is, however, not generally felt in the earliest stages, and may be imperceptible later from its small size; from the pylorus lying under the liver; from its being concealed by much distension of the bowels, or by ascites; or finally, because the tumour is situate on the posterior wall of the stomach. If no tumour is discoverable cancer may be confounded with chronic gastritis, or ulcer of the stomach; or purely neuralgic pains may be thought to be due to cancer. In *gastritis* the disease may have originated in imprudent diet; the pain and vomiting are more or less intimately related to diet; the appetite is often good, or even excessive; the tongue is furred, and headache, malaise, &c., are present. Cancer, on the other hand, arises independently of previous gastric troubles, and the pain and vomiting are less depend on food; finally, indeed, the pain becomes continuous. tongue may remain clean, but appetite is soon lost, and nutrition of the patient suffers. In gastritis also judicious tre ment materially or entirely relieves the symptoms, which cancer are but little, or only for a time, influenced.

The same important difference in the result of treatment is be noted between *ulcer* and cancer: ulcer is nearly always impro by proper dietetic treatment, whereas cancer may be scarcely all relieved. Ulcer also has more localised pain, and the p and vomiting are aggravated or brought on by food. Prof hæmorrhage is much more probably the result of ulcer, and oft occurs early; in cancer it appears late, if at all; coffee-grou vomit is seen in both. Anæmia is mostly the result of hæm rhage in ulcer, but in cancer it develops when the bleeding been slight or absent. A cicatrised ulcer may lead to troub some pains, but there is the long history, and the strength health are fairly maintained. Dilatation of the stomach occ very late in ulcer, but much more rapidly in cancer. Exc tionally the tumour of cancer may be simulated by the matti and adhesion of parts caused by ulcer (p. 610). The age to wh cancer is almost strictly limited, and the short duration of disease, are also important elements in its diagnosis.

In purely *nervous* affections of the stomach the pain is c tinuous, but there is generally no wasting.

Occasionally the anæmia of cancer has been so marked, the local symptoms so slight as to have led to the suspicion of *idiopathic* or *pernicious anæmia;* this is especially likely wh the tumour is small, or not easily reached. In such ca repeated examinations of the abdomen must be made, with p cautions previously indicated (p. 583).

Free hydrochloric acid is generally absent from the contents the stomach, but this is not peculiar to cancer. On the ot hand the presence of free hydrochloric acid on repeated exami tions is an argument against it. Lactic and other acids are lik to be present from dilatation, and fermentation of the conten and it has been even stated that the only condition under wh lactic acid (not introduced, preformed, with the food) can found in the stomach is the growth of cancer.

Prognosis.—This is very unfavourable, but perhaps less now that cancer of the stomach has come within the range practicable surgery. But it often happens that by the time tumour is first felt, and when the symptoms are scarcely eno to alarm, the lymphatic glands have become involved, and chance of benefit from operation no longer exists.

Treatment.—The diet must be arranged on somewhat differ principles from those followed in ulcer. Since the symptoms not solely dependent on food, and the tendency to exhaustion is so certain and pronounced, we must avoid the treatment by abstinence which is justifiable in the more curable disease. Only rarely, when the pain is very severe, may the patient be fed by enema for a few days; but generally the food must be of a light, nutritious kind, and must be given in the natural way—by the mouth. Milk, milk and soda-water, or milk peptonised with liquor pancreaticus, may be given in some cases; in others it may be thickened with farinaceous food, or eggs; or more solid nutriment may be borne, such as fish, chicken, or oysters. Wine of a light kind may be allowed; champagne is often useful on account of its effervescence, and in late stages brandy may be necessary. The symptoms of pain and vomiting must be dealt with much as in ulcer. For vomiting, ice internally, either alone or with milk, and ice applications to the epigastrium; effervescing medicines, small quantities of iced champagne, extract of opium in doses of $\frac{1}{6}$ to $\frac{1}{4}$ grain, or morphia in pill or solution ($\frac{1}{8}$ to $\frac{1}{6}$ grain) should be tried. Opium or morphia is again constantly required for the pain; or locally a small blister or chloroform liniment will sometimes give relief. Constipation must be met by enemata, or by saline purgatives, or by pills of aloes and iron.

If dilatation is a prominent symptom, and large quantities of food are vomited every few days, much relief is often afforded by washing out the stomach daily with the stomach-pump or syphon-tube (*see* Dilatation).

The following operations have been performed for cancer of the stomach: *Pylorectomy*, or resection of the pylorus; this is only possible in the earliest stage and has rarely been successful. *Gastro-enterostomy*, in which the duodenum, or other portion of the small intestine lower down, is joined to the stomach at a point where the wall is healthy, so as to create a gastro-enteric fistula by which the chyme may pass from the stomach into the alimentary canal below it. *Enterostomy*, or *Jejunostomy*, by which a portion of the small intestine is attached to the abdominal surface, and the patient is fed through a fistula made in it. Even *Œsophago-duodenostomy* has been successfully performed, the entire stomach being removed.

For fibrous stenosis of the pylorus, Loreta's operation of mechanical dilatation has been performed. An incision is made into the stomach, a finger of each hand is introduced into the pylorus, and the stricture is forcibly widened.

DILATATION OF THE STOMACH.

Acute Dilatation.

It occasionally happens that the stomach becomes rapidly and enormously dilated. In one of two cases described by Fagge, seven pints of a greenish fluid were removed from the stomach during life. The previous symptoms were pain and vomiting, which may have indicated an acute or sub-acute gastric catarrh, and the patients, with the occurrence of the distension, became collapsed and died. As the stomach from which so much fluid was taken had contracted to its natural size of death, Fagge suggested the name *acute paralytic distension.*

The following are the physical characters given by him :—(1) A rapidly increasing distension of the abdomen which is unsymmetrical, the left hypochondrium being full, while the right is comparatively flattened. (2) The presence of a surface-marking which descends obliquely from the left hypochondrium towards the umbilicus, and which corresponds with the lesser curvature of the stomach. This seems to move up and down each time the patient breathes. (3) Dulness and fluctuation in the pubic region with resonance over the front of the abdomen. (4) The production of a splashing sound on manipulation.

The **Treatment** is obviously the removal of the contents of the stomach as promptly as possible by means of the stomach-pump or syphon-tube.

Chronic Dilatation.

Chronic dilatation of the stomach results from (1) various conditions which produce obstruction of the pylorus; and from (2) conditions which alter the contractile power of the muscular walls. The causes of obstruction are most often cancer; cicatrices of ulcers less commonly; very rarely pressure from without, binding down by adhesions, or dragging of a prolapsed kidney; and, quite exceptionally, cicatrices from corrosive substances, which, however, generally involve the œsophageal aperture.

The causes of weakening of the muscular tissue are chronic inflammation (gastritis), excessive overloading of the stomach, such as sometimes occurs in the insane, in drinkers and in gluttons, excessive formation of gas from whatever cause arising, and the interference with its nutrition which occurs in prolonged fevers and anæmia. Obstruction produces the greatest extent of dilatation.

Physical Signs of Dilatation.—In marked cases, when the abdomen is exposed, it is seen to be asymmetrical, presenting a rounded *prominence* in its left half. This prominence extends below the level of the umbilicus, its lower margin having a curve convex downwards and outwards, from the lower part of the costal margin to the right of the middle line. The left half of the epigastrium may be sunken above another shorter curved line, which corresponds to the lesser curvature of the dilated stomach. From time to time a wave of *peristaltic movement* passes from left to right and downwards across the prominent part. A portion at the extreme left, about the size of the hand, quickly forms a convex prominence, with a decided amount of resistance to pressure; in a few seconds the swelling subsides, and another part, more to the right, swells up for a similar length of time. After each successive portion of the stomach-wall has become hard and prominent the whole subsides. This phenomenon occurs spontaneously, or may be set up by manipulating the abdominal wall, or flicking it with the finger sharply, or sometimes on mere exposure of the abdomen. Percussion of the swelling gives varying results, according to the proportions of air and liquid which the dilated stomach contains; generally the lower part is dull, the upper part is tympanitic; and some change in the relative positions of the two sounds may be obtained by altering the position of the patient, the dull area always being the lower.

By sharp movements of the stomach, as when the patient is shaken, or the prominent stomach is roughly manipulated (best by suddenly pressing upon it, and quickly withdrawing the hand) the liquid contents are set in motion, and *splashing* can be heard and felt.

Other tests have been employed; thus, if half a drachm of sodium bicarbonate and the same quantity of tartaric acid be introduced separately, but within a short interval into the stomach, carbonic acid will be set free, which will give some clue as to the position of the organ. Penzoldt pumps in a quart of water, and observes what difference it makes in the areas of dulness and resonance. Leube introduces a long sound through the mouth into the stomach, and pushes the point against the anterior wall of the abdomen. In health a sound should not go in more than twenty-four inches.

A characteristic feature of dilatation is the manner in which vomiting takes place. The food is retained very well for three or four days, and then a large quantity, two or three pints, is vomited at once. It is generally of a greyish-brown colour, frothing on the surface; and on microscopic examination it shows numerous spores of the yeast-plant, *torula cerevisiæ*, and the spores, in groups of four, known as *sarcina ventriculi*, so called from their resemblance to boxes or bales tied round with a cord.

In addition to the vomiting, the patient suffers from discomfort or actual pain, which is increased as the contents accumulate, and is temporarily relieved after they are evacuated. Great thirst, loss of strength, emaciation, pallor, and constipation are also observed. Much mental depression, and sometimes tetany and convulsions also occur. The urine is scanty, and may contain acetone or acetic acid.

Diagnosis.—This depends upon the physical characters above detailed, of which visible peristalsis is the most conclusive, percussion and splashing probably least so, from the proximity of the colon.

Prognosis.—Dilatation from narrowing of the pylorus must persist as long as the disease causing obstruction; and treatment other than surgical can only be palliative. When the distension results from weakening of the muscular walls, the outlook is more favourable, and recovery may take place.

Treatment.—For dilatation of the stomach, the operation of washing it out has been found of great value. The over-distended organ is thus relieved of the accumulation of liquid and undigested food; and any catarrh that may co-exist is at the same time benefited. The operation may be done with the stomach-pump; or a tube introduced into the stomach may be connected with a funnel, and the stomach is filled by raising the funnel above the level of the mouth and pouring in water; it is emptied again by depressing the funnel, and inverting it into a suitable vessel. Or the tube in the mouth may be connected by a Y-shaped joint with one tube passing upwards to a funnel, or other receiver, and another downwards into a vessel; when water is poured in, the lower tube is closed by the fingers just below the joint, and when it is desired to empty the stomach the upper tube is compressed and the lower left free. These two syphon arrangements have the advantage that they can be used by the patient himself, although they do not empty the stomach so completely as the stomach-pump.

Whatever apparatus is used, the stomach is first emptied entirely of its contents, and is then rinsed out, one or two pints of water being introduced and removed, and the process being repeated till the contents come out nearly clear. The water used for washing it is either pure, or contains bicarbonate of sodium (one or two per cent.), or salicylic acid (one per cent.). The washing should be done once daily, half-an-hour before the largest meal.

Food should be given in small quantities and at short intervals. Starchy and saccharine foods should be restricted or even prohibited to prevent fermentation; and liquids should not be given during digestion, or even at all. To compensate for this, they may be injected into th[illegible] Tender meats, meat essences, minced meats, and a little fat or cream may be given. The bowels should be kept active, if necessary, by salines, such as magnesium sulphate and Carlsbad salts.

DISEASES OF THE INTESTINE.

ENTERITIS.

There are several conditions, affecting different parts of the alimentary canal, which may properly be termed enteritis, or inflammation of the intestine. For instance, the catarrhal process, of which some forms of diarrhœa are the result; tubercular and typhoid ulcerations in the ileum; the ulcerative inflammation of the colon, known as dysentery; and the acute changes set up by intussusception and strangulations, are all, in fact, enteritis. But a large number of these have already received distinctive names; others are only secondary conditions, which produce few symptoms beyond those of the primary disorder; and in others, again, inflammation of the coats of the intestine involves a peritonitis, which throws the symptoms due to the mucous inflammation completely into the shade. Thus the number of cases which require separate description as enteritis is but a small one, though it is probable that a fair consideration of the pathological side of many of our intestinal cases, such as diarrhœa, would show that the name might be justly used more often.

The following forms of enteritis will be here described:—*catarrhal enteritis*, *English cholera*, *infantile enteritis*, *sprue*, *diphtheritic enteritis*, *phlegmonous enteritis*, *mucous colitis*, and *ulcerative enteritis* and *colitis*.

CATARRHAL ENTERITIS.

(*Intestinal Catarrh.*)

Anything which irritates the mucous membrane of the intestine may set up catarrh—such as unsuitable food, certain poisons, and purgative drugs. It is also ascribed sometimes to chill; but a much more potent factor in its production is excessive heat, and it is always more prevalent in the hot weather of summer and autumn than in the remaining part of the year. This frequency in the summer concerns people of all ages, but infants are especially attacked. It is not at all clear what is the immediate cause which the heat brings into play. In very hot weather food is more difficult to keep fresh, and milk turns with great readiness, so that the chances of an infant having an unsuitable or irritating food are much increased. Micro-organisms are probably often involved in the process, and many kinds of bacteria have been found in the intestine in health and disease. The two most constantly present are the *bacterium lactis aerogenes* in the upper part of the intestine, and the *bacillus coli communis* in the lower. Catarrh of the intestines may also arise from passive congestion in cardiac and hepatic disease.

Anatomy.—The changes in the mucous membrane of the intestine are similar to those in other parts of the body. The tissues are more vascular, and become swollen, and large quantities of mucus are excreted. The epithelial cells of Lieberkühn's glands are swollen, cloudy, and become detached, and cellular infiltration takes place in the intertubular tissue. In more advanced cases the solitary follicles are enlarged, and they may become eroded, and produce small ulcers (*follicular enteritis*). In some cases, also, ulcerations occur in other parts of the mucous membrane, and the secretions may consist of muco-pus, or even pus. As a rule, the inflammation subsides, but it may lapse into a *chronic* condition, with more prominent changes in the mucous membrane. Sometimes there is considerable thickening, with slaty discoloration of the surface; often—especially in the chronic catarrh of infants—there is atrophy of the mucous membrane, involving the glandular layer, but leaving the muscular layer of the mucous membrane, and the submucous tissue, intact.

Symptoms.—The chief symptom of enteritis is diarrhœa, or the frequent passage of motions, loose or liquid in consistence. This symptom is due, not only to the alteration in the secretions poured into the intestinal canal, but also, and largely, to the increased peristaltic movements which the irritation of the mucous membrane calls forth. The condition of the fæces varies much: they are generally at first abundant, liquid, and brownish in colour, with flakes or lumps of more solid matter; but they soon become paler, or, it may be, yellowish or sometimes green; in consistence they are often quite watery, or perhaps slimy, or they contain lumps of brownish mucus. Under the microscope there are particles of undigested food, meat fibre, starch granules, and fat, with crystals of ammonio-magnesian phosphate, epithelial and pus cells, and bacteria. The bowels may be moved from two or three times a day, to ten, twelve, or more.

Pain is often present, but not so much continuously as in the form of colicky or griping attacks, which precede the passage of the motions and subside again afterwards. Actual tenderness is not generally present, nor does examination of the abdomen reveal anything characteristic. Gurgling noises and *borborygmi* from time to time accompany the more active intestinal movements. The temperature is variable: it may be raised one or two degrees, or remain normal. Frequently the appetite is lost, the patient complains of thirst, the mouth is dry, the tongue is slightly furred, and a considerable degree of bodily weakness results when the diarrhœa is excessive. A very sudden and acute attack may begin with vomiting.

In most cases the symptoms pass off in the course of a few days; the diarrhœa may cease suddenly, leaving a long interval before the bowels are again opened, or the motions may gradually become less frequent, gaining a firmer and firmer consistence. If the complaint becomes chronic, the patient is troubled with three or four evacuations daily of watery mucus, with occasional griping pains. The imperfect digestion and absorption of food may lead to considerable loss of nutrition.

Some differences may be noted in the symptoms, according as one or other part of the alimentary canal is affected. Thus catarrh of the *small* intestine is more likely to be present if the stomach is at the same time involved; it is less likely to be accompanied by diarrhœa, which must depend finally upon the action of the large intestine. The evacuations often contain bile and undigested food, and mucus, if present, is more intimately mixed with the fæces. In catarrh of the *large* intestine, the mucus occurs in separate masses; muco-pus or pus itself may be recognisable. As catarrh approaches the *rectum*, tenesmus is more likely to be a symptom.

Treatment.—The patient should remain in bed and be kept warm. Beyond this, in slighter cases, a careful regulation of the diet may be all that is necessary. Gruel, arrowroot, beef-tea, or mutton broth, with rusk or toast, milk and soda-water, or milk and lime-water in small quantities at a time, should take the place of the ordinary meals. They should not be given too hot. Many cases, however, will require drugs in addition. If it is quite certain that irritating matters, such as unripe fruit, are the cause of the attack, and are still in the bowel, a laxative may be given in order to get rid of them and so prevent further irritation. For this purpose a single dose of castor oil may suffice, or one of compound rhubarb powder, or of calomel. But generally, by the time the case comes under treatment, there has been a free evacuation, and it is desirable to check the excessive peristalsis and abundant discharge, as well as to relieve pain. The tincture of opium may be given in doses of 5 minims every four hours, combined with an astringent such as hæmatoxylum, catechu, the aromatic chalk powder, or dilute sulphuric acid. Bismuth is also of value, and may be given with opium. If the griping is very severe, morphia may be injected subcutaneously. If the diarrhœa is obstinate and exhausting, an enema of two ounces of starch containing 15 minims of laudanum may often be used with success. In children, opium must be used as little as possible; bismuth, and aromatic chalk powder, must first be tried. If opium be necessary, the dose for a child one year old should not exceed one minim of the tincture, and it is well to begin with less.

In chronic intestinal catarrh, rest, careful regulation of the diet, with opium and astringents, are still required. Here also enemata may be found useful—two or three pints of warm water containing 1 per cent. of boric acid, salicylic acid, tannin, or acetate of lead.

English Cholera.

Cholera Nostras.—This is an acute gastro-enteritis, which occurs occasionally in the summer months, and appears often to be set up by unsuitable ingesta, such as sausages, meat pies, shell-fish, or food in a state of decomposition. The patient is seized with vomiting and diarrhœa; the evacuations soon have a rice-water appearance, and serious collapse, with cold surface, small pulse, sunken eyes, and cramps in the calves, ensues. The pathology of these cases is not clear. There is reason to believe that some of these cases, and some described as acute enteritis, are instances of poisoning by ptomaïnes, but the subject requires much investigation yet. *Ptomaïnes*, or *cadaveric alkaloids*, are poisonous alkaloidal bases, which have been found in decomposing meat, fish, gelatine, cheese, yeast, and putrid corpses; several have been isolated and chemically analysed. On the other hand, micro-organisms may play their part. A comma-bacillus (*spirillum Finkleri*) has been found in some cases of cholera nostras, and Klein found bacilli in some fatal cases of wholesale poisoning by meat, the symptoms being diarrhœa, vomiting, abdominal pain, languor, giddiness, and fainting.

Infants are frequently seized with a somewhat similar affection (*cholera infantum*) in which vomiting and frequent diarrhœa quickly bring them to a dangerous state of collapse, with depressed fontanelle, and almost complete unconsciousness.

The principles of **Treatment** are the same: extreme care with the diet, and the use of astringents; but a preliminary purge is here out of the question, and we must have recourse from the first to stimulants, such as brandy, ether, and ammonia. For infants, bismuth or aromatic chalk powder, with Dover's powder, in very small doses; for adults the tincture of opium in 5 or 10 minim doses, with ether, or aromatic spirits of ammonia.

Infantile Enteritis.

Children are subject to a chronic intestinal disorder, which is probably, in part at least, of a catarrhal nature. It is by far most frequent in infants who cannot be brought up at the breast, or must be weaned early and have to depend on artificial feeding. Sometimes immediately after birth, or directly upon weaning, in other cases after a longer or shorter interval, the food disagrees; the child vomits, and the motions are loose; or with not much vomiting, there is constant diarrhœa. If it is taking milk, this is returned in curdled lumps; by the bowels also particles of undigested milk are passed, but the fæces, which in the healthy infant are yellow, generally become grass green in colour, are slimy from mucus, and very offensive. Among the poorer people, children under twelve or fifteen months are often fed with potato and meat, much the same as their parents, and in such cases undigested particles of these foods are found in the motions. The children suffer from griping pains, which are brought on by the ingestion of food, or occur just before a loose evacuation. Frequently the abdomen is distended with flatus, but presents nothing to examination with the hand; the condition, though often spoken of by the parents as "consumption of the bowels," has no necessary connection with either tubercular ulceration, tubercular peritonitis, or caseation of the mesenteric glands (*tabes mesenterica*). After a certain time the child emaciates, lies fretful in its mother's arms, and is constantly whining, or screaming from time to time with sudden pain. Finally, collapse and death may terminate the scene.

Morbid Anatomy.—In most cases the appearances in the intestine are confined to enlargement of the solitary follicles, with perhaps abrasion and ulceration; in old cases the intestines may be atrophied. Both small and large bowel may be involved. Rickets, or tubercular and caseating glands, if present, must be regarded as secondary or independent.

Treatment.—The management of the child's food is the first consideration. If the child is suckled, it should be seen that the mother's milk is of good quality, and that it is not given too frequently; a baby of one or two months old should not be nursed more than once every two or three hours, and the mother must carefully abstain from giving it the breast simply because it cries. If from any cause the mother is unable to suckle it, the most efficient substitute is a wet-nurse, and if this cannot be provided, recourse must be had to the milk of domestic animals—namely, the ass, goat, and cow. Such milk differs from human milk in its composition and its digestibility, the latter property depending on the kind of coagulum which the casein forms in the child's stomach. Analyses show that animal milk differs from human milk in the following points—Cows' milk contains more proteids, more salts, only slightly more fat, and less lactose, or sugar of milk: the casein coagulates in large lumps. Goats' milk is very similar to cows' milk, but contains more fat: the casein coagulates in large masses. Asses' milk is deficient in proteids and fat, but contains as much sugar: the casein clots i small particles like that of human milk. Asses' milk is, therefore, often well borne, and digested with ease, but bulk for bulk it is not sufficiently nutritious to be continued for long. Moreover, the greater facilities for obtaining cows' milk will always be a reason for using it, if it can be so modified as to approximate its composition and properties to that of human milk. The addition of water will reduce the proportion of the proteids and the salts, but the mixture must then be sweetened with sugar, and a little cream may with advantage be added. For infants under one month old there should be one part of pure milk to two parts of water, for those a little older up to four months there may be equal parts of milk and water, and after that age two parts of milk to one of water. The dilution also slightly influences the coagulation of the casein; but this is rendered still more like that of human milk by the addition of lime-water. This addition, moreover, neutralises the acidity, which distinguishes cows' milk from that of women. The lime-water should be in the proportion of one part to two parts of the pure milk employed. The same purpose is served by adding barley-water instead of pure water. Another mode of getting over the difficulty of the digestibility of the casein is to peptonise the milk by means of Benger's liquor pancreaticus. This may suffice for a time, but the objection is that it is doing the stomach's work entirely, and depriving it of the exercise of its own proper function. The above method of preparing milk by the addition of water, a little sugar, and liquor calcis, if somewhat rough, is at least in numerous cases satisfactory.

But more accurate methods of compounding a suitable milk have been devised, and should be preferred, or resorted to at once if the above should fail. Artificial human milks are either direct mixture of cows' milk, cream, lactose, and water in certain proportions; or they consist of cows' milk from which some portion of the casein has been removed. Coulier* gives the following:—Cows' milk, 300 grammes; water, 339·5 grm.; cream, 13 grm.; lactose, 15 grm.; calcium phosphate, 1·5 grm. Rotch uses a mixture of:—Milk, 1 ounce; cream containing 20 per cent. of fat, diluted with one-third part water, 2 ounces; milk sugar, 3⅜ drachms in water, 3 ounces; lime-water, diluted with three-fourth parts water, 2 ounces. Frankland's artificial milk is as follows:—Heat half a pint of skimmed cows' milk to 35° (95° F.); add rennet. After ten to fifteen minutes break up the curd finely, strain the whey off and boil it, adding 110 grains of lactose. Strain again, and add to it two-thirds of a pint of fresh cows' milk, and then two teaspoonfuls of cream. This should be made every twelve hours. Cheadle gives similar directions:—The cream is to be removed by skimming, after the milk has stood some time. The remainder is to be divided into two equal portions, from one of which the casein is to be removed by rennet. The other half is then added to the whey, and the whole of the cream added. For an older child, the casein may be removed from a third part only, as in Frankland's recipe. He points out that the casein remains the same in respect of its coagulating property.

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* Halliburton's "Chemical Physiology and Pathology."

But cow's milk may be in transmission readily contaminated with organic matters of a bacterial nature. This serious fault is to be corrected by *sterilisation*—that is, either by boiling or steaming the milk before use. Boiling has long been employed with this object, but it has the disadvantage of removing a certain proportion of the proteids by coagulation of albumen, which, with some fat, is removed in the scum; and boiled milk is also less palatable. In the different forms of *sterilisers* the milk is not boiled, but it is heated just short of boiling, either by exposure in bottles to steam for a certain time, or by the immersion of a vessel containing the milk in a larger vessel containing water, which is then boiled. The bottles are loosely plugged while exposed to heat, and tightly stopped when the process is complete. A kitchen steamer may be used, the milk being placed in suitable bottles. Steaming for twenty minutes will suffice for milk to be used immediately, but if it is required to keep for the day a longer time must be given. Rotch directs that his mixture shall be sterilised before the lime-water is added. Sterilisation in one form or another is especially desirable when the infant is suffering from summer diarrhœa; it has another advantage, in that it diminishes the risk of infection by typhoid fever, scarlatina, and diphtheria, when those diseases are prevalent. "Humanised" milk can be obtained which is both sterilised and assimilated to the normal composition.

If the child is fed with the bottle, it is essential that this should be kept scrupulously clean in all its parts, and washed thoroughly and scalded after every feeding.

Sometimes it is necessary to stop all milk food for a time, and small quantities of raw meat juice, slightly sweetened (a teaspoonful every four or six hours), or Brand's essence, or diluted Valentine's meat juice may be supplied instead.

Medicinally, very small doses of mercury may be given with advantage—for instance, calomel, $\frac{1}{8}$ or $\frac{1}{6}$ grain with sugar, or hydrarg. c. cret., $\frac{1}{2}$ grain or 1 grain, with 1 or 2 grains of bicarbonate of sodium, two or three times a day. This, with the improved diet, will often be sufficient; but if diarrhœa is obstinate and associated with much pain, it is desirable to give bismuth (2 to 5 grains of the nitrate) or astringents, such as aromatic chalk powder (5 grains), extract of hæmatoxylum (2 grains), or tincture of catechu (10 or 15 minims). Either of these may be combined for older children with small doses of opiates in the form of Dover's powder (½ or 2 grains) or tincture (1 minim to a child one year old).

Brandy must be given if there is much collapse in doses of 10 or 30 minims every three or four hours.

SPRUE.

Sprue, or psilosis, is a form of chronic catarrhal enteritis which occurs in the tropics. The patient has soreness of the mouth, flatulent distension of the abdomen, and looseness of the bowels. The tongue, palate, lips and buccal mucous membrane are much reddened, with superficial erosions, patches of congestion, and minute vesicles or apathæ. Saliva dribbles from the mouth, and eating and swallowing are painful. The stools are copious, pultaceous, white or greyish, acid, with a disagreeable smell, different from the normal fæcal odour, and containing numerous bacteria. Temporary attacks of acute diarrhœa with watery stools also occur. In fatal cases the intestine has been found much thinned and atrophied, with different degrees of erosion and ulceration of the mucous membrane, and degeneration of villi and glands and follicles. The disease is generally cured by an absolute milk diet, which must be continued for many weeks after the last symptom, otherwise a relapse may take place.

DIPHTHERITIC OR PELLICULAR ENTERITIS.

The mucous membrane of the intestine is generally much injected, and presents numerous white patches of membrane. These lie usually along the edges of the valvulæ conniventes, leaving free the depressions between the valvulæ; but they may be more extensive. This form is seen sometimes as a complication in other diseases—*e.g.*, purpura rheumatica and Bright's disease. The symptoms may be masked by those of the general disease, or may be similar to those of catarrhal enteritis.

PHLEGMONOUS ENTERITIS.

In this form all the coats of the bowel are involved, including the serous coat or peritoneum. There is generally intense redness and vascularity, the mucous and submucous coats are thickened, softer, and more friable than natural, and the peritoneum is vascular, sticky, or covered with lymph. It is usually a local disorder, arising from mechanical interference with the bowel or its circulation, or from the spread of inflammation from adjacent parts. Thus in intussusception, and in strangulated internal and external hernia, the bowel involved is affected with enteritis; in obstruction from other causes, also, the bowel above becomes distended from retained fæces, and ultimately its walls inflame, numerous ulcers may form, known as *distension ulcers*, and frequently the bowel ruptures. Cases are, however, recorded of localised enteritis involving all the coats of the bowel, in which none of these mechanical causes were discoverable. The **Symptoms** are often, for the most part, the result of the accompanying peritonitis, and consist of pain, vomiting, local tenderness, collapse, distension of the abdomen, and febrile reaction. Diarrhœa is not commonly present; even if the mechanical conditions allowed it, the implication of the muscular coat of the bowel will lead to its paralysis and inaction. A fatal result often ensues. **Treatment** must be directed to the primary cause, and in its absence is practically the same as that of peritonitis.

Mucous Colitis.

In this peculiar affection of the lower bowel large pieces of membrane, or *casts*, are discharged from the rectum from time to time. It occurs most frequently in females, but it is a rare disease. There is commonly troublesome constipation, and other symptoms of chronic dyspepsia, and the discharge of membrane is preceded by severe griping pains. The casts may be several inches or even feet in length, and an eighth to a quarter of an inch in thickness; they are yellowish brown, transparent, and gelatinous. They consist of mucus, in which are embedded cylindrical epithelium, some round cells, and crystals of cholesterin and ammonio-magnesian phosphate. The disease lasts for years, and is very little influenced by drugs or diet. It has been relieved in some severe cases by opening the colon in the right loin.

Ulcerative Enteritis and Colitis.

Apart from the specific lesions of typhoid fever and tuberculosis, the follicular ulcerations mentioned under enteritis, and the perforating ulcer of the duodenum, the small intestine is little liable to ulceration. In the large intestine, however, extensive ulcers may occur, which are not follicular, nor of a specific dysenteric nature; and in this kind of ulceration, the small intestine is sometimes involved. The ulcers are often large, irregular, confluent, and extend to the muscular coat; sloughs may be found adhering to the surface, and the intervening mucous membrane is vascular, swollen, or pigmented. Sometimes the ulcers perforate.

The **Symptoms** of ulcerative colitis are paroxysmal griping pain and diarrhœa, with slimy, offensive, fluid motions, containing blood not mixed with the fæces, but no great quantity of mucus. Sometimes shreds or sloughs are present. The patients become sallow, have irregular pyrexia, frequently vomit, lose strength and flesh, and die from exhaustion or perforation. It is a disease of middle age, about equally common in the two sexes (Hale White).

Treatment should be similar to that of diarrhœa—rest, bland nutritious diet, astringents, and opium.

PERFORATING ULCER OF THE DUODENUM.

Ulcers occur in the duodenum of the same nature and under similar conditions with those that affect the stomach. They are, however, much more rare. In their course and progress they present no essential differences; they cause similar symptoms, and, like gastric ulcers, may give rise to hæmatemesis or perforation, contract adhesions to surrounding parts, or cicatrise and narrow the channel of the duodenum. They are more often latent than gastric ulcers, the pain is situate in the right hypochondrium, and may occur three or four hours after food. The **Treatment** is the same.

APPENDICITIS.

(*Perityphlitis.*)

Although the anatomical relations of the cæcum appear to offer unusual facilities for the accumulation of fæcal matters in this part of the bowel, and for inflammation of its walls as a result, it is clear, from *post-mortem* evidence, that the great majority of the cases hitherto described as typhlitis and perityphilitis arise from lesions of the appendix cæci. Only very rarely does the perforation of the cæcum by a tubercular or stercoral ulcer (*see* p. 646), or a foreign body lead to this local form of peritonitis.

Ætiology.—Appendicitis is much more frequent in early life than in middle or old age, and in the male than in the female sex. It often appears to be determined by indigestion, or the use of indigestible foods, and occasionally by cold, or by injury.

Pathology.—The alleged proximate causes are as follows:—(1) Some solid body in the cavity of the appendix, irritating and inflaming its coats. This may be a cherry-stone, orange-pip, seed, bristle, or similar substance; but more often it is a concretion, the size of a pea or plum-stone, yellow or greyish in colour, which consists of dry fæcal matter, mixed with mucus, and hardened by the deposit of lime-salts. The frequency of this cause (about 25 per cent.) is less than was once believed. (2) Catarrh of the cæcum spreading to the opening of the appendix, which is thereby obstructed, so that the cavity is distended by retained secretions. (3) Torsion of the appendix and strangulation of its vessels by undue distension of the cæcum. (4) Micro-organisms, especially the *bacillus coli communis.* This is constantly present in the large intestine. Its influence is probably only exerted when the mucous membrane of the appendix is already inflamed or ulcerated. (5) Very rarely a tubercular ulcer may be the origin of the disease.

The results are very various. In the appendix itself may be noted infiltration, thickening of its coats, distension of its cavity with catarrhal products or pus, suppuration of its substance, and finally ulceration and gangrene. In most of these conditions the trouble spreads almost necessarily to the peritoneal covering, and in a certain proportion of cases to the sub-peritoneal connective tissue. The peritonitis thus arising is mostly localised and adhesive, matting the appendix to the adjacent bowel, and forming thus more or less definite resistant masses in the right iliac region. If the connective tissue is involved it will be also infiltrated, and assist in the production of the swelling. In either case, the inflammatory products may be absorbed, or may suppurate: in the latter case, the abscess, if untouched by the surgeon, may burst externally through the skin, or discharge itself into the cæcum, bladder, vagina, rectum, or peritoneal cavity, its course being probably determined to some extent by the anatomical position of the appendix in the subject of the disease.

The appendix sometimes perforates or sloughs before adhesive peritonitis has occurred, and then a general peritonitis, of a very fatal kind, is rapidly determined. Occasionally, a peritonitis, at first local, gradually extends with the formation of peritoneal abscesses in different parts of the abdomen, until death takes place in from three to six weeks.

Symptoms.—The onset is often somewhat sudden. Spontaneously, or after some such indiscretion as has been referred to, the patient is taken with severe abdominal pain, at first diffused over the abdomen, but soon more pronounced in the right iliac fossa, with malaise, nausea, and some febrile reaction. The tongue is furred, the appetite fails, there is thirst, and the bowels are constipated; there may be also vomiting. The abdomen may be somewhat distended, but is generally rigid; and there is tenderness in the right iliac fossa. This tenderness is often definitely situate at a point about three inches from the right anterior superior spinous process, on a line drawn from this process to the umbilicus—*McBurney's point;* and in this neighbourhood, after a time, a certain amount of resistance can be felt.

These symptoms may continue for a few days, the vomiting, pain, and fever gradually diminish under treatment, and the trouble may subside in a few days. In many cases, however, beginning like this, and in others in which the onset is more gradual, the localised resistance becomes a definite tumour, situate above Poupart's ligament, extending from the anterior superior spinous process of the ilium to the pubes, and internally half or two-thirds of the distance from Poupart's ligament to the umbilicus. It is often quite dull on percussion, and sometimes it has a modified tympanitic note; the rest of the abdomen is supple and resonant. The temperature may rise to [illegible] or 104°, the pulse to 100 or 120. The pain may be irregular or paroxysmal, and often shoots down the right leg.

In its further progress the tumour generally subsides, gradually becoming less tender and smaller, so that it disappears in from ten to twenty days from the time it was discovered, while the fever and other constitutional signs diminish. On the other hand, the swelling may suppurate, with still further enlargement, increasing pain, tenderness, and illness, oscillating temperature with or without rigors, profuse sweating, and all the other indications of septic absorption. Fluctuation is then generally felt, and occasionally the tumour becomes resonant on percussion, from decomposition and formation of gas in the interior. Spontaneous recovery may even now take place by rupture of the sac into the alimentary canal, when the pain and discomfort are quickly relieved, and a quantity of pus may be noticed to pass by the rectum. Generally, however, the abscess has to be opened by the surgeon, and the convalescence may be slow, as often the abscess is a deep one, and there is much surrounding infiltration.

Rupture may take place into the peritoneal cavity, into the bowel, vagina, or other cavity. In the first case the symptoms of general peritonitis will succeed, in the others the swelling in the right iliac fossa will subside, pus is discharged with the stools, or *per vaginam*, and the patient gradually recovers.

Second and third attacks of appendicitis frequently occur in the same patient. Not only may fresh inflammation be easily set up by indiscretions in food or in locomotion during convalescence, but at intervals of months or years, either the existence of the foreign body, or a repetition of the exciting cause, may lead to a fresh attack of a similar nature (*relapsing appendicitis, relapsing typhlitis*).

In cases of early *sloughing* of the appendix, there may be the same local symptoms for a day or two, and then the signs of general peritonitis rapidly develop, which will be described later; or the local indications may be entirely absent, or so slight that they are scarcely noticed by the patient, and the case is from the first one of general peri

Diagnosis.—Appendicitis with limited peritonitis is at once recognisable from the position of the symptoms, from the nausea or vomiting, constipation, and fever. The leg is not drawn up as in psoas abscess. A very protracted case of appendicitis with much infiltration, in a middle-aged person, may give rise to suspicions of malignant disease; but disease of the appendix is very much more frequent in younger people.

Treatment.—The chief object should be to give complete rest to the intestine, so that the inflammation may be confined within the limits it has already reached, and no risk may be run of an extravasation of intestinal contents into the general peritoneal cavity. Especially, purgatives should be avoided, and opium should be given freely—for instance, 1 grain in pill, or 15 minims of the tincture every four hours. The diet should consist mainly of milk in small quantities, and nausea or vomiting may be allayed by sucking ice. Severe pain may also be relieved by leeches, and by linseed-meal poultices or hot fomentations, applied constantly. With regard to surgical operations, early intervention for the removal of an appendix at the first sign of its being involved is of more doubtful expediency, since quite 90 per cent. of cases recover under medical treatment, but probably more harm has been done by delay than by too early interference, and if the case does not go on favourably, if tumour and fever persist, if prostration occur, and the pulse fails, it is better to explore at once, evacuate pus and remove the appendix. If fluctuation occurs in the mass the course is plain, but pus may form behind the cæcum, and escape recognition. The swelling or tenderness of such an abscess may sometimes be detected *per rectum*. Relapsing appendicitis may sometimes properly be treated by the removal of the appendix in a third or later attack.

TUBERCLE, CANCER, AND SYPHILIS OF THE INTESTINE.

Tubercle.—Tuberculosis of the intestine has been already referred to under phthisis, but it may occur independently, especially in children, in whom the infection takes place probably through infected milk. In either case the lesions occur chiefly in Peyer's patches in the ileum, and in the solitary follicles of the ileum and colon. The process is the same as elsewhere, cell proliferation, caseation, necrosis, and ulceration; and the ulcers in the ileum may be of great extent, being round or oval, and frequently running transversely round the gut, rather than along it. The surface is irregular, with thickened edges, and the serous surface corresponding to it generally presents small white tubercles in some number. The symptoms associated with tubercular ulceration of the bowel are pyrexia and diarrhœa. The motions are generally abundant, pultaceous, fatty, and yellow in colour. Sometimes they are more fluid, and still yellow, and if the abdomen is distended there may be a close resemblance to typhoid fever. Hæmorrhage and perforation are rare. The treatment has been indicated (*see* p. 471).

Fistula in ano is no doubt sometimes tubercular in origin.

Cancer.—This is so often the cause of intestinal obstruction that it is not necessary to say much of it here (*see* Intestinal Obstruction). Cancer of the rectum, which occurs chiefly in elderly people, may be alluded to as it is liable to be mistaken for dysentery, the symptoms being local pain, straining or tenesmus, and the passage of small quantities of mucus, sometimes stained with blood. An examination with the finger will enable one at once to recognise the mass of new growth blocking the passage not far from the anus, and yielding the above-mentioned fluid.

Syphilis.—It is very rarely that syphilis affects the alimentary canal between the pharynx and the rectum; but it is an occasional cause of stricture in this last situation. Gummata form in the submucous tissue, and slowly lead to cicatricial contraction. They occur commonly in women, and little may be known of them till symptoms of stricture are observed, and the constriction is recognised by digital examination.

DIARRHŒA.

By diarrhœa we mean the passage of motions more often, and of looser consistence, than is normal. Much that can be said of diarrhœa has been described under the head of Enteritis, of which diarrhœa is a frequent symptom; but as this disorder is set up either by excessive peristaltic action, or by increase of the intestinal secretions, as well as by a morbid condition of the mucous membrane, it may arise in many other ways besides enteritis; and it is not always easy to say whether a catarrh of the bowel has any share in its production or not.

Causation.—A purely nervous influence may cause diarrhœa, as, for instance, fear; and a chill may produce it, possibly in the same way. The more common causes are irritating foods and liquids, impurities in the drinking-water or in the air that we breathe—causes that have already been mentioned in connection with intestinal catarrh. The production of diarrhœa is also illustrated by the use of purgative and laxative medicines, some of which act by exciting the muscular fibre, others by stimulating the intestinal glands, and others by actually inflaming the coats of the bowel. Besides the conditions described as enteritis, we have other changes in the bowel that are accompanied by diarrhœa. These are typhoid and tubercular ulcerations, which affect the lower end of the ileum, and perhaps the cæcum; dysentery, already described, which affects the colon and cæcum and sometimes the lowest part of the ileum; lardaceous disease, which invades both small and large intestines in cases of chronic suppuration and chronic syphilis, but most frequently in phthisis, where it is associated often with tubercular ulceration. Another condition of the bowel, which is accompanied with diarrhœa, is *lymphosarcoma*, which grows in the walls of the intestine, and there may attain a thickness of half or three-quarters of an inch, while the bowel itself, instead of being contracted by the growth, may be actually enlarged to a circumference of ten or twelve inches. It is of very rare occurrence.

Diarrhœa also occurs in some general illnesses where it is not so simply explained, as in septicæmia sometimes; and occasionally it accompanies the termination of a pneumonia or other acute fever by crisis. It must not be forgotten that frequent discharge of liquids in small quantities does not necessarily show that the canal of the bowel is quite free; thus intussusception which partly obstructs the gut is accompanied by the passage of mucus and blood; fæcal fluid mixed with mucus may pass very large masses of impacted fæces; and lastly, even a distinctly contracted intestine may allow some of the thin liquid which collects above the obstruction to pass through and simulate a diarrhœa.

Varieties.—Diarrhœa has received different names according to the nature of the matters passed; thus we have *choleraic* diarrhœa, in which the stools are profuse and watery, or like the rice-water stools of cholera; *dysenteric* diarrhœa, in which mucus is largely present, and perhaps blood; *lienteric* diarrhœa, or *lienteria*, the passage of undigested food; *bilious* diarrhœa, where the discharges are deeply stained of brown or greenish-brown colour, which is due, not so much to any increase in the quantity of bile secreted, as to the fact that the contents of the duodenum and jejunum stained with bile have been hurried through the alimentary canal, without giving time for the natural reabsorption of the bile pigment. *Colliquative* diarrhœa is a term applied to the profuse, exhausting, and intractable discharges, which occur in the last stages of phthisis.

Treatment. — This must depend upon the cause, or the associated condition. The treatment of cases resulting from enteritis has been already described, and the same principles may be followed in most acute cases. A critical diarrhœa may generally be left to itself, and some caution must be exercised in checking those which result from the congestive catarrh of heart and lung disease, or take place in Bright's disease. The diarrhœa of enteric fever should be left alone unless excessive, when starch and opium enemata are usefully employed. But in chronic and obstinate cases astringents become necessary, and of these several combinations are in favour. Opium is of the greatest value in quieting the movements of the intestines, and it may be combined with the direct astringents; of these hæmatoxylum, catechu, compound kino powder, hamamelis tincture, chalk, either as mistura cretæ or aromatic powder, bismuth, sulphate of copper, or dilute sulphuric acid are most used. In some cases with frothy or yeasty motions, where fermentation is obviously proceeding, antiseptics often do good, such as sulphurous acid in ½-drachm doses, salicylic acid, salicylate of bismuth (5 grains), or wood charcoal.

HÆMORRHAGE FROM THE BOWEL.

The passage of blood *per rectum* has already been noticed as occurring in enteric fever, in ulcer of the stomach, and in dysentery; it may, of course, happen with any considerable breach of surface, but simple oozing without obvious rupture may also lead to considerable hæmorrhages. The way in which the blood is passed may give a clue as to the point whence it comes. In bleeding from gastric or duodenal ulcers the blood is considerably altered by the secretions, and forms a black, tarry, semi-liquid, or treacly mass (*melæna*); in hæmorrhage from typhoid ulcers the blood is equally unmixed with fæces, but brighter red and more fluid than in the former case, from the action of the alkaline contents; the blood in dysentery is in streaks or small clots mixed up with mucus or pus, or thin fæcal matter, though from time to time small quantities of pure blood may be passed. Large quantities of blood may be lost from piles, or from an ulcer of the rectum. Here the bleeding is generally caused by the act of defæcation, the blood either streaking one side of the solid fæcal mass, or coming more or less pure in drops or streams after the motion is evacuated. In scorbutic, purpuric, and hæmorrhagic conditions (scurvy, purpura hæmorrhagica, acute yellow atrophy of the liver, malignant variola) blood comes from the rectum more or less mixed with fæces, or pure, according to the part of the intestine yielding it, or the freedom with which it escapes. The **Treatment** of hæmorrhage is described with the various diseases which may cause it.

CONSTIPATION.

The healthy action of the bowels depends on a sufficient supply of food, the waste of which forms the material for the fæces; a natural secretion of intestinal juices; and an intestinal muscular system readily stimulated and strong enough to force on the fæces from point to point. This action, however, varies in different individuals, who may still all be healthy. Most persons have an action of the bowels once a day, but others twice a day, and some only every other day. In disease this regular action is readily disturbed. Constipation, or retention of the fæces for longer periods than is natural, is a marked symptom at the onset of acute febrile diseases, in many gastric disorders, especially where vomiting is frequent, and in cerebral and spinal diseases, where the innervation of the bowel is no doubt influenced; for instance, constipation is often present in cases of cerebral tumour. But many persons, otherwise in good health, suffer from "habitual constipation."

Symptoms.—If left to themselves the bowels only act at intervals of two, three, four, or more days; the rectum becomes loaded with hard round masses of fæcal matter (scybala), generally rather pale, which are welded together into masses. The desire to go to stool perhaps at first only results in ineffectual straining efforts; but finally some scybala are passed, and the same may be repeated two or three times within a few hours, till the lower bowel is emptied. After this the bowel is inactive for another period of several days. During the retention the patient may suffer various inconveniences. Locally, there may be a sense of fulness in the perineum, and the hæmorrhoidal veins may swell. Sometimes there is pain down the thigh from pressure of the fæcal masses on the nerves in the pelvis; moderate distension of the abdomen often occurs, with perhaps flatulence and eructations, the tongue is often furred, whitish or dirty brown, and the breath may be foul. Some patients feel languid, confused, wanting in vigour or freshness, and have actual headache (attributed by some to the absorption of ptomaïnes), or even a great deal of mental depression. But it must be noted that often the more habitual the constipation, the less is the general disturbance; and many are not conscious of having anything the matter with them, though their last evacuation was many days before.

It is generally believed that, normally, when the fæces reach the top of the rectum, they excite a peristalsis by which they are promptly discharged, without any lengthened stay in the rectum; but it is certain that in habitual constipation, either from loss of sensitiveness or lessened power, the rectum tolerates the presence of fæces, and becomes enormously dilated to accommodate them. The scybalous condition of the fæces is explained by their slow progress along the large intestine, during which there is time for the absorption of most of the liquid contained in them; while they are moulded into shape either in the tube of the colon or in its sacculi. The possibility of scybalous masses in the rectum allowing some fæcal fluid to escape, or exciting a flow of mucus and thus simulating diarrhœa must not be forgotten.

Ætiology.—Habitual constipation is sometimes hereditary but it is much more often under the control of the individual being induced by sedentary habits, by indulgence in a too dry diet or in too much animal food, by repressing the desire to defæcate, or ignoring the sensation which precedes it. A false modesty in large households or in schools leads many young girls to postpone the call to empty the bowels, and lays the foundation for a sometimes quite serious trouble. Mechanical causes also play a part in habitual constipation; for instance, in women, a retroflexed uterus or a uterine or ovarian tumour. A growth in the wall of the lower bowel itself is often a cause of constipation for some time before it becomes a recognised obstruction.

Treatment.—For many cases of troublesome constipation much may be done without having recourse to drugs. The patient should make a regular daily visit to the closet, whether he feels any desire or no at the time; the diet should be modified so as to include a sufficiency of vegetables, fruit, fresh or preserved, or salad with salad oil; brown bread, whole meal bread, or oatmeal sometimes supplies the desired stimulus to the bowel. The diet also, should be liquid enough; and with some a daily evacuation is ensured by taking a tumblerful of cold water before breakfast To those of sedentary habits, walking exercise, horse-riding, or driving, is often of benefit.

But with all this it may be still necessary to have recourse to drugs, and a careful selection is requisite. As a rule, very active or drastic purgatives must be avoided; they produce abundant liquid motions, from the effect of which the intestinal muscle is completely exhausted, and consequently no further evacuation takes place for days afterwards. But it has already been shown that constipation depends on weakness of peristaltic action, and hence over-stimulation and exhaustion are especially to be avoided From this point of view much advantage is gained by combining with the ordinary laxatives those drugs which have a tonic effect upon the muscle. Such is especially nux vomica; and iron acts in a similar way.

The remedies more commonly employed are the saline mineral waters, as Friedrichshall, Püllna, Hunyàdi János (containing the sulphates of magnesium and sodium), and Carlsbad (mainly sulphate of sodium); the patient may take from a wineglassful to half a tumblerful before breakfast. Carlsbad salts, extracted from the water of the different springs, of which the Sprudel seems the most efficient, may also be given—a teaspoonful is dissolved in half a tumblerful of hot water, and drunk before breakfast. Another laxative that has been largely used of late is the cascara sagrada; it is perhaps less likely than some other vegetable laxatives to exhaust the bowel; it may be given every night in doses of 30 or 40 minims of the liquid extract, combined with syrup of ginger. A useful combination is that of extract of aloes with extract of nux vomica, one grain or a grain and a half of the former with a quarter or half a grain of the latter, given in the morning before breakfast; sometimes extract of belladonna, one-sixth or a quarter of a grain, may be also added.

Fagge speaks highly of a method suggested by Dr. Kent Spender; and I can confirm this recommendation. He orders pills containing a grain of sulphate of iron with one or one and a half grain of extract of aloes or of compound rhubarb pill. One of these is given three times in the day after meals; but active purgation must be avoided, and directly this seems likely to be produced, the three pills must be reduced to two, and if necessary to one, each day, and, indeed, this result generally follows. Whereas at first three pills daily are required to keep the bowels regular, after a time two suffice, later on one only, and ultimately the bowels act without any assistance whatever. Nux vomica and belladonna may be also combined with aloes and iron. Galvanism, massage, and kneading of the abdomen are means which may be resorted to in exceptional cases. Repeated enemata of cold water are sometimes advised, but they are apt to do harm by distending the bowel unduly; for an occasional evacuation the injection into the rectum of a drachm of glycerine is often useful.

INTESTINAL COLIC.

The term colic, though obviously derived from the word colon, means a spasmodic abdominal pain, or a pain presumably due to contraction of visceral muscular fibres. These fibres may be those of the ureter (renal colic), of the bile ducts (biliary or hepatic colic), or of the intestines (intestinal colic).

Ætiology.—The most frequent cause of intestinal colic is irritating and unsuitable ingesta, such as pork, cheese, high game, shell-fish, ices, &c. In children, colic is a common result of indigestible food, or even simple excess. With these may be classed the more active purgatives. On the other hand constipation is often associated with colic, and this is markedly so in the colic due to lead poisoning, whether acute or chronic (*see* Lead Poisoning). Some cases may perhaps be referred to a purely nervous source, for instance, the severe pain of gastric crisis in locomotor ataxy. Lastly, mechanical and acute inflammatory lesions of the bowel, such as strangulation and intussusception, lead to severe pains, which are partly or wholly due to muscular contraction. The term colic is, however, generally reserved for conditions in which there is no structural or inflammatory change.

Symptoms.—The important symptom is pain, which is situate about the umbilicus, but may move about other parts of the abdomen. This pain is often relieved by pressure, but sometimes there is tenderness. The abdomen is either drawn in, and the abdominal muscles are contracted, or the belly is distended from the presence of flatus. When flatus is present borborygmi are produced by its movements, as it is driven on by the intestinal spasm.

The pain may be so severe as to cause much collapse, with profuse clammy sweat, and small feeble pulse. There may or may not be vomiting: often there is constipation; on the other hand, some ingesta, which cause colic, set up active diarrhœa with brown watery stools, and mucus after a time. Here the colic is associated with a definite though slight enteritis. The more active purgatives also produce griping and "colicky" pains, which are commonly diminished after each evacuation.

Diagnosis.—This is not always simple, for colic of a purely functional kind may be confounded with the pain of peritonitis or that of acute intestinal obstruction. In other words, a severe spasmodic abdominal pain may, as already stated, be due to serious disease, as well as to a temporary and easily curable disturbance. Lead colic is often mistaken for intestinal obstruction, when a glance at the gums would give the right clue. On the whole, it is the absence of the more positive indications of peritonitis or obstruction, and of the facial signs of abdominal disease, the relation to unsuitable foods, or the fact that the patient has had similar attacks before, which will help most to make the case clear.

Treatment.—Obviously, cases of severe abdominal pain must be treated with much caution. If the pain is certainly due to irritating ingesta, relief generally follows the exhibition of purgatives, such as an ounce of castor oil with 15 minims of tr. opii, or half an ounce of magnesium sulphate with half a drachm of tr. hyoscyami, or 5 grains of calomel; and a similar line of treatment is used in lead colic (*see* Lead Poisoning). A warm-water or castor-oil enema may also help; and hot fomentations or a hot-water tin should be applied to the abdomen. If there is any likelihood that peritonitis or obstruction may be present, it is better to treat the case with anodynes, such as one grain of opium internally, repeated in two or three hours, or the subcutaneous injection of morphine; while the external applications are also used. The successful result, if it is colic, will not be materially delayed, while the injury that a purgative may do is avoided.

INTESTINAL OBSTRUCTION.

The intestine may be obstructed in several ways, which may be first briefly enumerated:—

1. Foreign bodies, large gall-stones, or collections of fæcal matter in its interior.
2. Intussusception or invagination.
3. Changes in the intestinal walls, such as strictures caused by healed ulcers, or by malignant growths.
4. Volvulus.
5. Strangulation by bands or through apertures.
6. Diminution of the calibre due to traction on the intestine, or to compression from outside in various ways.

Pathology.—*Foreign Bodies.*—Among the foreign bodies found obstructing the bowel are fruit-stones, pebbles, coins, bullets, pins, needles, hooks, and false teeth. Sometimes large masses are formed of vegetable fibre, wool, or husks of oats, matted together. It is especially in lunatics that foreign bodies of this kind are found. Occasionally a large gall-stone is the cause of a fatal obstruction; or it passes *per anum* after more or less difficulty. Such gall-stones may be two or three inches in length by three or four in circumference; they are formed in the gall-bladder, and reach the bowel, not through the bile-duct but by ulceration through the walls of the gall-bladder and the duodenum. They commonly obstruct the small intestine, especially the lower part of the ileum or the duodenum. Fæcal masses may accumulate in the same way as has been described under Constipation, and form an insuperable obstacle, in the rectum, sigmoid, or colon. They are more common in women than in men, and are mostly met with in adults. Masses of magnesium salts, after the extreme use of carbonate of magnesium, have been known to form in the bowel and give rise to obstruction.

Intussusception.—This presents special features which make it desirable to consider it separately (*see* p. 652).

Strictures.—These occur both in the small and large intestines; they arise either from contraction of cicatrices of ulcers, or from new growths in the intestinal walls. Of the different forms of ulceration, it appears that dysenteric and catarrhal ulcers most frequently give rise to stenosis, and that typhoid rarely, if ever, does so. But in many cases of stricture there is no evidence as to what form of ulceration has preceded it. Simple strictures are generally single. Occasionally, they follow the reduction of a strangulated hernia, or injuries to the abdomen. The new growths causing stricture are, as a rule, of malignant nature, and usually *cylindrical epitheliomata*. In the majority of cases it is a primary growth, but occasionally it is secondary or extends from adjacent parts. The form it commonly assumes is that of a band or ring round the bowel, by which the internal circumference is considerably reduced, even to the size of a cedar pencil, but rarely completely obliterated. The longitudinal extent of the growth is often not more than one or two inches; the inner surface is frequently ulcerated. Simple tumours, such as *adenomata* and *fibromata*, are only occasionally the cause of intestinal obstruction.

Strictures, whether simple or malignant, are more common in the large than in the small intestine; this is especially true of malignant disease. And in the large intestine itself the sigmoid flexure is most often the seat of stricture; the descending colon comes next in frequency, and the hepatic flexure is somewhat more often attacked than the splenic flexure. In nearly 70 per cent. it is the sigmoid or descending colon. Females are somewhat more often affected than males; the patients are generally middle-aged, malignant cases being usually about forty, and cases of simple stricture somewhat younger. Strictures are essentially of slow development, and the gradually increasing obstruction influences the condition of the bowel above. This becomes distended by the accumulation of fæces, and hypertrophied in its efforts to force its contents past the stricture. Fæcal matter naturally accumulates, but is from time to time passed through, sometimes after symptoms of almost fatal intensity; occasionally, however, foreign bodies, such as fruit-stones, of larger diameter than the aperture, may collect above it. Increasing distension and pressure from within may ultimately lead to ulceration, gangrene, and perforation of the gut above the stricture. It is important to recognise that, in strictures of the sigmoid or descending colon, fæcal matter often accumulates in the cæcum.

Volvulus.—By this term is meant the twisting of a loop of bowel upon itself, so that the two portions at the ends of the loop cross and strangulate each other. It is most common in the sigmoid flexure, which may form a loop sufficiently free, from the length of its meso-colon, for the purpose. More rarely it happens that the cæcum is twisted on its vertical axis so as to cause obstruction, or bent up in front of itself; and occasionally the small intestine forms a volvulus of the same kind as that described in the sigmoid.

Another form of volvulus is seen, in which two loops help to strangulate one another; so, for instance, the sigmoid flexure may be doubled round a loop of the ileum, or two loops of the ileum round one another. Volvulus occurs in males more often than in females, and, as a rule, between the ages of forty and sixty.

Strangulation by Bands and through Apertures.—This class of cases is precisely analogous to ordinary cases of hernia, and they are often described as internal strangulation and internal hernia; a loop of intestine, commonly the ileum, slips through an aperture, and is strangulated by the margin of the aperture grasping its neck. Such apertures may be slits in the omentum or mesentary, but are more often formed by a band of adhesion stretching from one part of the abdomen to another, under which the loop of gut passes, or by the same band forming more or less complicated loops in which the gut is involved. Bands of this kind arise from a former peritonitis, probably local in extent, and often in connection with the pelvic viscera or the appendix cæci. Such bands are often solitary, though they may be accompanied by other adhesions not forming bands. The pedicle of an ovarian tumour may strangulate the bowel.

A frequent cause of this form of obstruction is the congenital abnormality known as *Meckel's diverticulum.* This forms a finger-like projection from the unattached side of the ileum, from two to four inches in length, and half to three-quarters of an inch in diameter. It has the same serous, muscular, and mucous coats as the ileum, and is a remnant of the omphalo-mesenteric duct, by which the primitive alimentary canal communicates with the yelk-sac. It arises from the ileum, at a point eighteen to twenty-four inches from the cæcum, and its blind termination is generally free; but it may be attached by a fibrous band to the anterior abdominal wall at the umbilicus, or to the mesentery, or to the peritoneal surface at some other point. A ring is thus formed, through which a loop of gut may slip, and then become strangulated.

When once the loop has slipped through, strangulation is favoured by everything which increases the contents of the loop, such as more air or intestinal liquid; and not infrequently the loop itself becomes twisted like a volvulus.

Strangulation by bands and apertures is more frequent in males than in females, and occurs at all ages, but in greatest number between the ages of twenty and forty. Among cases occurring in early life, strangulation by Meckel's diverticulum is the most common.

In many instances some history of previous peritoneal trouble may be obtained.

Compression and Traction.—The last class of cases is called "contractions" by Fagge, "compression and traction" by Bristowe, and "anomalous forms of obstruction due to isolated bands and to adhesions" by Treves. The last writer distinguishes them as strangulation over a band; acute kinking due to traction upon an isolated band, or an adherent diverticulum; adhesions retaining the bowel in a bent position; adhesions compressing the gut; matting together of several coils; changes effected in the intestinal coils due to simple traction; and narrowing of the bowel from shrinking of the mesentery after inflammation. The cases are comparatively rare; they concern the large intestine and small intestine with about equal frequency; and they are likely to be preceded by a history of peritonitis.

Effects of Obstruction upon the Bowel.—In a fatal case of acute obstruction of the intestine, the bowel above the seat of obstruction is found enormously distended, while that below is collapsed and empty. The distension begins immediately above the constriction, and affects the bowel for a greater or less distance, according to the severity or duration of the obstruction. Thus, in obstruction at the sigmoid, the whole colon and much of the small intestine are affected; in obstruction of the ileum, the small intestine is distended and the colon collapsed. In the upper distended portion is a quantity of fæcal matter, light brown or yellowish-brown in colour, and of uniform thick liquid consistence; and this is the same, whether the obstruction is in the small or large intestine: there is never sufficient absorption by the intestinal vessels to form the harder and drier fæces of health. In chronic cases the distended bowel becomes gradually hypertrophied from its efforts to overcome the obstruction. If this is unrelieved, ulceration, sloughing, and rupture, or perforation takes place, with peritonitis as a result. In acute strangulation, sloughing may occur at the seat of constriction, from the direct interference with the circulation; in the chronic obstruction of strictures the bowel yields in the distended portion above. Where large fæcal accumulations are the cause of obstruction, the scybalous masses irritate the mucous membrane, and set up catarrh and ulceration, forming so-called *stercoral ulcers*.

General Symptoms of Obstruction.—The symptoms of intestinal obstruction are vomiting, constipation, pain, and distension of the abdomen. The special feature of the *vomiting* is its *stercoraceous* or fæcal character. At first the contents of the stomach are discharged, and subsequently bilious matter; but comparatively early in acute cases, and with the final obstruction in chronic cases, the vomited matter consists of light or dark brown thick liquid, with a distinct or even powerful fæcal odour. This was at one time explained by supposing that the obstruction set up an *antiperistalsis*—*i.e.*, a peristaltic movement in the wall of the bowel in an upward direction, contrary to that of health; but it is now commonly allowed that the ordinary downward peristalsis is sufficient to cause the occurrence of fæcal vomiting, the liquid next to the intestinal wall being moved downwards, while a central current is established in a reverse direction—that is, towards the stomach.

The *pain* of obstruction is variable. In acute cases it is very severe, generally paroxysmal at first, and not becoming continuous, as pointed out by Treves, until the obstruction is complete. Its situation is sometimes determined by the position of the lesion in the abdomen, but often it is referred to the umbilical region, though the strangulation may be in quite another part of the abdomen. The pain in chronic obstruction may be very slight, but aggravated when obstruction increases to a marked degree. Tenderness is not generally present until peritonitis sets in.

Constipation is an important feature in obstruction, though not in itself conclusive, as it is present in other conditions. It is often absolute from the time of obstruction—not only is there no motion, but also no flatus whatever. Occasionally, however, the lower bowel may contain fæces at the time the obstruction occurs, and these may be discharged, or removed by an enema.

But a more complete description must be given of the symptoms and course of intestinal obstruction; and we must distinguish between acute cases, of which the strangulation by a band is the most typical example, and chronic cases, of which malignant stricture of the sigmoid is the best instance.

Symptoms of Acute Obstruction.—In a case of strangulation by a band, the patient is seized with intense pain in the abdomen, generally in the neighbourhood of the umbilicus; he may be walking about, or having a meal, or he may be awakened from sleep. Sometimes the attack is attributed to a strain, or to some unaccustomed or indigestible food taken some hours previously; but it is often impossible to prove the connection. The patient then vomits, either directly or within a short time, the vomited matter being the contents of the stomach. The pain is almost continuous, and vomiting is excited by every attempt to take food. The abdomen generally becomes tense, but the actual distension varies with the position of the obstruction; if this is in the upper part of the small intestine, the abdomen may be flat, or distended only at the upper part, above the umbilicus; if the lower part of the ileum is strangulated, the abdomen is uniformly enlarged. Neither motion nor flatus is passed *per anum;* and the vomiting, at first gastric, then bilious, becomes ultimately stercoraceous.

The effect upon the patient is very grave. Collapse soon sets in; the face is drawn, the eyes are dark and sunken, the pulse small and quick, the temperature normal or subnormal, and much flesh may be lost in a few days. The tongue is dry, and there is constant thirst. The urine is scanty and high-coloured; its quantity tends to be less, the higher the seat of the obstruction—a fact which is to be attributed to the generally more constant vomiting, so that but little food is absorbed into the system. If the condition is unrelieved death supervenes, either from exhaustion, or from the occurrence of peritonitis, of which a general diffused tenderness may be the chief indication. The duration of the case is from four to six days.

The forms of intestinal obstruction which commonly cause acute obstruction, besides strangulation by bands and apertures, are intussusception, volvulus, impaction by gall-stone, and some forms of acute kinking by adhesions. Some differences may be noted. In volvulus of the colon the onset is sudden, and there is severe pain, though not generally so severe as in strangulation by bands; and it is at first less continuous. The intestines become extremely distended very early, and local tenderness over the distended coil, as well as rigidity of the abdominal wall, are noticeable. Tenesmus occurs in a smaller number of cases. Strangulation over a band and acute kinking produce acute symptoms like those above described, but the pain is generally less continuous, the case is less rapid, and the symptoms more variable in intensity. In cases of complete obstruction by gall-stones, the onset is usually sudden, the pain severe and continuous with exacerbations. Vomiting appears early, is abundant, and becomes stercoraceous. There may be a history of previous illnesses attributed to gall-stones. Other foreign bodies are less often the cause of acute obstruction.

Symptoms of Chronic Obstruction.—In chronic obstruction such as is due to malignant disease of the sigmoid flexure or of the descending colon, the symptoms are at first only indicative of a moderate interference with the passage of fæces; there is some local pain, and occasional vomiting, not particularly related to the ingestion of food. Constipation occurs irregularly, but it can be overcome by aperients. From time to time the constipation is very troublesome, vomiting is more frequent, yet not stercoraceous, the abdomen becomes greatly distended, and the hypertrophied coils become visible in peristaltic movements on the surface of the abdomen. When the distension mainly affects the colon, as in the case of sigmoid cancer, the transverse colon bends downwards in the middle and forms two enormous vertical coils. When the small intestine is chiefly distended, and the colon is collapsed, the distended coils often lie transversely across the abdomen. With the peristaltic movement can be heard gurgling sounds, or borborygmi.

After a week or ten days of such symptoms, some fluid motions may pass, and then quickly several large evacuations of liquid fæces, by which the abdomen is rapidly reduced to its normal capacity, and all the symptoms are relieved. This sequence of events may recur more than once, but in some such attack the constipation becomes complete, nothing is passed *per anum*, vomiting is more frequent and becomes stercoraceous, the abdomen is enormously distended, with visible moving coils, there is severe pain of griping character, and, after some days, it may be as many as ten or twelve, death takes place from exhaustion, or from rupture of the bowel and peritonitis. If the case is seen early, a tumour can be sometimes detected in the left iliac fossa, but its recognition may be quite impossible when the abdomen has become much distended. The patient may also present the loss of colour and emaciation so common in cases of malignant disease.

The chronic cases which produce somewhat similar symptoms are other forms of stricture of the large intestine, strictures and growths of the small intestine, most forms of compression, traction, and matting of the gut by adhesion, compression of the gut by tumours outside it, some cases of impaction of a foreign body, and fæcal accumulations.

Sufferers from *fæcal accumulations* have generally had previous attacks of constipation, which have only been relieved by strong aperients; and at length even these are useless. The patient then has indigestion and flatulence; the abdomen swells, it may be to an enormous extent, and causes dyspnœa by its pressure on the diaphragm, while the mass of fæces may press upon the lumbar or sacral plexus, or the abdominal or pelvic veins. Sometimes the fæces excite catarrh of the bowel, and a little thin fluid escapes, which may be mistaken for a genuine relief. Nausea, eructations, and vomiting follow, and the distension of the coils may be so great as to be visible on the surface. Occasionally the vomiting becomes stercoraceous, and death takes place from exhaustion. In many cases a tumour due to the accumulated fæces can be felt: this is especially the case if the obstruction is in the large intestine, when the mass often occupies the cæcum and the ascending colon. The tumour is hard, uneven, rounded or elongated in shape, and generally painless. Sometimes it is not hard, but rather doughy in consistence. The duration of these cases may be several months.

Position of the Stricture.—The differences to be noted between strictures of the small intestine and those of the large are, that in the former vomiting occurs earlier, and is more determined by the ingestion of food: in the latter distension is greater, the proximity of the stricture or growth to the anus may lead to alterations in the shape of the motions, which may be ribbon-shaped; and tenesmus is frequently present. The stools, moreover, often contain blood. If the abdomen is distended, the prominence is greatest in the middle line in obstruction of the small intestine or of the ascending colon; but more general if the sigmoid or the descending colon is the seat of disease.

Diagnosis of Obstruction.—The chief points of distinction between the different varieties of intestinal obstruction have been already mentioned; but the diagnosis will often be difficult. The history must be carefully considered; the vomited matter must, if possible, be seen, as friends will often represent as fæcal what is merely gastric or bilious; and the rectum should be examined with the finger. In cases affecting the colon the attempt has often been made to ascertain the site of the obstruction by passing instruments, or injecting liquids into the bowel. But neither of these methods is trustworthy; the flexible tube will bend on itself long before it reaches the obstruction, and enemata will either distend the bowel enormously below the stricture, or possibly pass through the stricture, thus giving a wrong estimate as to the position of the lesion. Another very important point is the diagnosis of intestinal obstruction from *acute peritonitis* and some other acute lesions. Peritonitis resulting from inflammation of the appendix cæci is frequently confounded with acute strangulation. The patient is seized with pain in the abdomen, followed by vomiting and more or less distension. Constipation is often present for forty-eight hours or more, and during this time there may be the greatest difficulty in diagnosis. The following may serve as guides:—In peritonitis there is diffused tenderness and general distension; the temperature is often elevated; the vomiting is perhaps less severe than in strangulation, and rarely fæcal. The onset may have been preceded by symptoms pointing to the appendix; but other forms of peritonitis may also give rise to difficulty.

On the other hand, intestinal obstruction has sometimes been mistaken for other diseases—*cholera, lead colic, hepatic colic, renal colic, arsenical poisoning,* and even *meningitis.* In the latter case, recorded by Fagge, the abdomen was retracted in obstruction of the jejunum, and the patient was delirious and sick.

Treatment.—When the diagnosis of acute intestinal obstruction is established the operation of *laparotomy* or opening the abdomen should be performed without delay; and the cause should be ascertained and an attempt made to remove it, as, for instance, by reducing a strangulated coil, dividing a band, unfolding a volvulus, or extracting a gall-stone. Even if the diagnosis between obstruction and peritonitis cannot be determined, the same operation may be employed, as it is desirable in either case, assisting both diagnosis and treatment. In cases of obstruction, the operation often presents great difficulties, and it is well to do it as soon as possible, because the longer the duration of the case, the greater will be the distension of the bowel, the less the vitality of the tissues and of the patient, who may readily sink under a prolonged operation. If the obstruction cannot be found, or the parts are matted together so that it cannot be dealt with, the bowel must be opened above the obstruction either in the wound employed, or through a fresh opening in the loin as seems most expedient. If the patient is already so exhausted when the lesion is first recognised, as to make it highly probable that he will sink under a peritoneal operation, the bowels should be simply opened at the most distended part, and a fæcal fistula established. In suspected cases previous to operation, and in cases where for any reason an operation is not performed, the patient should be fed by nutrient enemata; and purgatives should be avoided, for except in one or two special cases, they can only aggravate the case by exciting the peristalsis of the intestines to fruitless efforts, whereby the congestion and strangulation of the bowel may be actually increased, and the pain and vomiting are rendered more severe. Much relief may be given by means of opium internally (half a grain to a grain every four hours) or morphia subcutaneously (one-sixth grain from time to time as the pain requires it). It must be remembered, however, that this treatment, while relieving the pain and checking sickness, removes two important symptoms, and may lull to a false security while the fatal mischief is progressing. Locally, relief may be furthered by the application of turpentine stupes; or of flannels wrung out of hot water, and sprinkled with tincture of belladonna, or opium; or of hot linseed-meal poultices.

Looking to the uncertainty of early diagnosis, and the difficulty of operating, Mr. Hutchinson advocates *abdominal taxis* under anæsthetics, in preference to laparotomy. The abdomen is thoroughly kneaded, and, if necessary, the patient is held with his head downwards, and shaken. Mr. Hutchinson urges that even in the cases of bands, the bowel may not be very tightly held, and may again be displaced by these means.

In *chronic* obstruction, which is chiefly the result of strictures and growths, whether in the small or large intestine, the diet must be carefully selected, with the object of ensuring regular digestion, and the easy passage of the intestinal contents through the constriction. Enemata, and occasionally laxatives, may be used to maintain a periodical evacuation. If an obstinate constipation ensues, and especially if great distension and sickness occur, the treatment must be assimilated to that of an acute obstruction. Opium may be given, with or without belladonna, while food must be given in only small quantities, or *per rectum*, when relief may be shortly obtained. Eventually, if life is to be prolonged, an operation will become necessary.

For stricture of the colon a colotomy should be done in the right or left loin, according to the position of the obstruction; in some cases the removal of the diseased portion of bowel (*colectomy*) may be desirable. For the small intestine, laparotomy will be probably required, and the bowel must be dealt with in various ways.

For fæcal accumulations, large and frequently repeated enemata generally suffice, but the case requires to be long under treatment by careful diet, exercise, electrical treatment, or massage, to restore the bowel to its former power.

INTUSSUSCEPTION.

If one segment, say a few inches, of the intestine slips into the portion immediately adjacent, it forms an *intussusception*, or *invagination*. It will be at once seen that this must present from without inwards to the centre of the bowel three layers of bowel-wall, of which the innermost may be called the *entering* layer; the outermost, the *receiving* layer; and the portion joining these two, the *middle* layer. The process of intussusception may continue, so that more and more bowel is involved, and this usually takes place by the entering and middle layer moving in uniformly together, and, as it were, dragging in the outer layer after them. In this way, as more of the entering layer disappears into the intussusception, the middle layer increases at the expense of the outer layer. The inner bend, between the entering and middle layers, remains always the same, the most advanced portion of the intussusception; the outer bend, between the middle and outer layers, is constantly shifting. It is clear that any portion of bowel might slip into a segment above, forming as *ascending* intussusception; or into the bowel below, forming a *descending* intussusception. It is with the latter that we practically always have to do.

Intussusceptions occur at any part of the bowel, and have received names accordingly; thus, those of the small intestine are called *enteric*, those of the large intestine *colic* or *rectal*. But at the point of junction with the ileum and the colon two varieties occur—(1) The *ileo-cæcal*, in which the ileum and cæcum pass into the ascending colon, the ileo-cæcal valve forming the most advanced point, the ileum the entering layer, and the cæcum the most advanced part of the middle layer; (2) the *ileo-colic*, in which the lowest part of the ileum is inverted through the ileo-cæcal valve—that is, an enteric intussusception continued into the colon. Of the different forms the ileo-colic is the rarest, and the ileo-cæcal is the most common, forming nearly half of all cases.

Very important changes, dependent on the anatomical relations of the intestines, ensue upon an intussusception. The intussusception, if at all extensive, forms a thick cylindrical swelling, partly from containing three layers of bowel all round instead of one, partly on account of the congestion and œdema to be presently explained. From the mesenteric connections of the bowel this cylinder has a curved shape, since the vessels which supply the inner and middle layers are of the same length as those supplying the receiving layer, and yet have not only to reach the border of the intussusception, but to go into its interior between the inner and middle layers, so that they drag upon the upper end of that part of the bowel. As the intussusception increases it moves farther along the gut, and the internal cylinder of an ileo-cæcal intussusception may even reach the rectum, and project from the anus. At the same time the tumour becomes larger. The disposition of the vessels just described leads to their compression and strangulation, and consequently to congestion and œdema of the walls of the intussusception; and even to hæmorrhage from the mucous surface, and the discharge of blood *per rectum*, a condition of the greatest value in diagnosis. If the case is not quickly fatal, inflammatory changes ensue in the layers of the bowel, binding them together, and interfering both with the further progress and with the reduction of the intussusception; and, lastly, from the strangulation of the blood-supply to the entering and middle layers, these may become gangrenous, slough off, and be discharged *per rectum*. If this has been preceded by the secure adhesive union of the entering layer to the angle between the outer and middle layers, the canal of the bowel is practically restored, and an actual cure may be the result, though very rarely; if the union is imperfect, the detachment of the inner cylinder may be followed by a fatal extravasation.

Ætiology.—The cause of intussusception is obscure in the majority of cases. Sometimes it has arisen after strains or direct injuries, or after unsuitable ingesta, or in connection with diarrhœa. Intestinal polypi and cancerous tumours have sometimes seemed to favour its occurrence. It may happen at all ages, but is much more frequent in children; and it affects males more often than females in early life, though the difference between the sexes is not so great in adults. Little that is definite can be said as to the immediate mechanism of intussusception, except that it is due to an irregular peristaltic action.

Symptoms.—The onset of an acute intussusception is not unlike that of strangulation by bands—that is, the patient is rather suddenly seized with pain, which is more or less constant, though aggravated from time to time, and griping in character. Nausea and vomiting also occur, but constipation is not generally present at first; on the other hand, the bowels are usually moved, and either thin fæces, or (what is especially characteristic of intussusception) *blood* with mucus is passed. Indeed, blood is passed *per rectum* in four-fifths of the acute cases; and often a certain amount of tenesmus is present. The abdomen is not always much swollen, and an examination reveals generally another characteristic feature—the presence of the *tumour* which results from the intussusception. Its position is of course related to the site of the lesion; in the more ordinary ileo-cæcal form it is at first situate in the right flank, but as the intussusception increases it is felt in the upper part of the abdomen, and is generally oval, cylindrical, or sausage-shaped, lying transversely across the abdomen above the umbilicus. Subsequently it passes into the splenic region, the left flank, and left iliac fossa, and ultimately can be felt by the finger in the rectum, or actually projects from the anus. Sometimes there is complete constipation, much distension, and fæculent vomiting; at others collapse sets in rapidly, and death takes place in twenty-four hours, or from two to five or six days. Death is especially rapid in quite young infants.

But the symptoms are not always so acute; indeed, an intussusception may exist for weeks or even months. In these more chronic cases the extent of bowel involved is generally less, and the canal is not completely obstructed. The bowels may thus be opened, though blood is passed at the same time in about half the cases. The patient suffers from paroxysmal griping pains, not necessarily of great severity. The abdomen is flaccid, and the characteristic tumour may be felt to vary in consistence; hardening simultaneously with the griping pains, but soon becoming soft, and even imperceptible when they subside.

The terminations of the subacute and the chronic cases are various; they may ultimately lead to death by exhaustion, or to complete obstruction with vomiting, constipation, abdominal distension, and visible coils; or they may set up a local peritonitis, followed by the formation of abscess, or by a more general peritonitis; or the intussuscepted portion may separate by sloughing, and so the intestinal canal may be re-established.

Diagnosis.—Spasmodic pain, vomiting, the passage of blood *per rectum*, and the presence of an elongated tumour which varies in consistence from moment to moment, and lies in the course of the colon, or occupies the rectum, are the chief features of intussusception. *Enteritis* and *dysenteric diarrhœa* in children may resemble it, but there is no tumour.

Treatment.—An *acute* intussusception should, as soon as possible, be met by an effort at reduction. This can, fortunately, be done in many instances by the comparatively simple expedient of injecting air, water, or oil into the rectum and colon, so as to force the intussusception backwards, and thus unfold or reduce it. Chloroform should be given, and a Lund's inflator or a suitable rectum tube, attached to a small bellows or bicycle pump, is inserted in the rectum, and the buttocks closed firmly upon the instrument. Air is then pumped in, and its progress along the bowel is watched by the hand placed on the abdomen, and the tumour formed by the intussusception may be felt moving towards the right side of the abdomen, and ultimately disappearing. Too much force may rupture the coats of the bowel, and for this reason air seems to me preferable to any liquid, since it is less unyielding, and yet can be renewed to any extent required. After the reduction, small doses of opium may be administered, with careful dieting for a few days. If inflation is unsuccessful, laparotomy should be performed, and the intussusception reduced, or, failing that, resected.

In *chronic* forms the diet must be carefully regulated, and opium may be given to relieve pain, spasm, or sickness. Here, also, inflation may be attempted, but in consequence of secondary changes it may be unsuccessful; and then the severer operation will be required.

INTESTINAL WORMS.

The following are the worms more commonly met with in the human alimentary canal:—

Cestoda, or tape worms . . (κεστός, a girdle)	*Tænia solium.* *Tænia mediocanellata.* *Bothriocephalus latus.*
Nematoda, or round worms . (νῆμα, a thread)	*Ascaris lumbricoides.* *Oxyuris vermicularis.* *Trichocephalus dispar.* *Anchylostomum duodenale.*

Trichina spiralis also develops in the alimentary canal, but its symptoms mainly result from its infesting the voluntary muscles.

It is essential to say something of the life-history of each of these worms before dealing with the symptoms which it produces, and the means to be employed for its expulsion.

TÆNIA SOLIUM.

Anatomy.—The Tænia solium is a flat, ribbon-shaped worm, very narrow at one end, broader at the other, from ten to twelve feet in length, and divided into a number of small segments. There is no alimentary tube, but two canals extend the whole length of the animal, constituting the so-called water-vascular system. At the narrow end is a globular swelling, or head, not larger than a pin's head, presenting a central prominence, or proboscis, surrounded by a row of twenty-six hooklets; and four suckers are placed at the sides. Below the head is a narrow portion or neck, where the segments are quite small and thin, but they gradually get broader and larger towards the other extremity. As they become larger, these segments acquire sexual characters, and are then called *proglottides*: each one bears male and female organs, the apertures for which are on one edge of each segment, alternately on the one side and the other of the tapeworm. A fully developed Tænia solium may contain about 850 segments, of which only the last 80 to 100 are mature. A mature proglottis measures half an inch in length, by a quarter of an inch in breadth. The uterus is an elongated cavity running the whole length of the segment, and giving off from seven to ten branches on each side, which again branch freely. The ova measure ·03 mm., are slightly oval in shape, and have a thick shell, presenting fine radiating lines under the microscope. The embryo develops while the ovum is still in the uterus.

The tapeworm inhabits the small intestine, being attached firmly to the mucous membrane by its head, while the chain of segments lies, partly coiled, along the bowel, as far down as the lower end of the ileum. As the lowest segments become mature they are detached, and are passed with the fæces. The ova may escape during this transit, or subsequently by the decomposition or rupture of the segments, and they thus become scattered on the ground, or on leaves, grass, or elsewhere.

For the further development of the ovum it is essential that it shall be taken into the stomach of an animal; and in the case of the Tænia solium it is the pig that performs this service, swallowing the ova with vegetables, or with the refuse and garbage that it feeds upon. Arrived in the stomach of the pig, the shell of the ovum is dissolved by the gastric juice, and the embryo, or *proscolex*, provided with six hooks, escapes, to bore its way into the gastric or intestinal vessels, and thus be carried to the liver, muscles, or other part of the body. In some such situation the embryo remains, and develops into a little globular bladder about the size of a pea, with which is connected, by a narrow segmented neck, a head with hooklets, suckers, and proboscis, precisely like that of the complete tænia. The head and neck, however, are usually retracted or inverted into the centre of the little cyst. These cysts occur in great numbers in the muscles of the pig, and the flesh so affected is described as "measly pork"; and they are seen occasionally in man, in the connective tissue, in the eye, and elsewhere, and have been known as the *cysticercus telæ cellulosæ*. In these situations they can develop no further, and in course of time perish; but when the flesh containing them is eaten by man, or a carnivorous animal, the head and neck are extended from the globular cyst, the cyst is dissolved in the stomach of the host, the head attaches itself by its suckers to the alimentary mucous membrane, and the segments of tænia successively grow upon it until a complete tapeworm (*strobila*) is formed.

Symptoms.—The presence of the worm may cause no symptoms at all, and it may only be recognised by the discovery of segments in the motions. Sometimes disagreeable sensations in the abdomen are described, or gnawing or colicky pains, irregularity of the bowels, and deficient or voracious appetite; itching at the nose or at the anus, salivation, and vomiting also occur. More remote symptoms are giddiness, fainting, and languor; headache, mental disturbance, depression, and even fits, either hysterical or epileptic in character. These are more likely to be aggravated in persons of hypochondriacal or hysterical tendencies. It is obvious that there is nothing pathognomonic in these symptoms, they can only give rise to a suspicion, which must be confirmed by the appearance of the segments. These the physician should himself see, since they might be simulated by fragments of mucus or half-digested food. It may be desirable in some cases to give a purgative to bring away segments.

This tænia may be distinguished from the others by attention to the characters above specified.

Treatment.—This should never be undertaken unless the presence of a tapeworm is absolutely certain. The method of treatment is the administration of a drug, which is fatal to the worm, and the subsequent removal of the worm by a purgative. In order that the former drug, or anthelmintic, may come into full contact with the worm it is desirable to have the intestines as empty as possible. In most cases it is sufficient for the patient to have nothing after six or seven in the evening, and to take the anthelmintic before breakfast the next morning. If the bowels have been previously confined, they may be cleared by a dose of castor oil the day before the morning dose. In either case, the anthelmintic should be followed in three or four hours by a dose of castor oil or compound rhubarb powder. Several drugs will destroy the tapeworm; the most commonly used in England is male fern, of which the liquid extract may be given in a dose of 1 to 1½ drachm suspended in mucilage, or emulsified with tincture of senega and water. Other remedies are kousso, in the form of infusion; oil of turpentine from ½ to 2 ounces, which should be followed by a purgative to ensure its not being absorbed; decoction of pomegranate root bark in three or four doses of 1 or 2 ounces each every half-hour; tannate of pelletierine, 8 to 12 grains in capsule; and kamala powder, 1 to 3 drachms in wine or water.

The dead worm must be looked for in the motions which follow this treatment, by mixing them with water, stirring, and pouring off the upper portions from time to time. The cure cannot be considered complete unless the head of the worm is found; for the worm may break at the neck, and if the head remains attached to the bowel, it will give rise to fresh segments, and ultimately to a complete tapeworm. In this case, a period of almost exactly three months elapses before the mature segments again appear in the fæces; and as it is obvious that the head may elude even a very careful search, it is quite as well not to repeat the treatment forthwith, but to wait until the reappearance of segments conclusively shows that there is still a worm in the bowel.

TÆNIA MEDIOCANELLATA VEL SAGINATA.

In addition to the pair of water-vascular canals present in the Tænia solium, this tapeworm possesses a third, occupying the middle line. Its head is provided with four suckers, but is without proboscis or hooklets. In length the animals may attain four yards, the segments number from 1200 to 1300. The complete development of the sexual organs occurs about the 600th segment; the last 150 to 200 are ripe proglottides. The mature segments measure three-quarters of an inch in length, by one-third in width, and the uterus extends the whole length of the segment, giving off on each side from twenty-five to thirty lateral branches, which divide at their extremities. The ova are only a little larger than those of Tænia solium, and have the same shape. The cysticercus is commonly found in beef or veal, and not in pork. The mature segments frequently find their way out of the anus, independent of the act of defæcation.

The **Symptoms** and **Treatment** are the same as those of the Tænia solium.

BOTHRIOCEPHALUS LATUS.

This worm is much larger and longer than either of the preceding, measuring from seventeen to twenty-six feet in length The head is elongated, and presents only two long suckers. Th segments are about 3000 in number; they measure in the middl half-an-inch broad, and only one-seventh of an inch in length but lower down they become more square in shape. The uteru is unbranched, but is bent several times upon itself. The ov measure ·07 mm. in length, and have a lid at one end. Th portions of the tapeworm that are detached are often severa feet in length. The life-history is similar to that of the tænia but the cysticerci inhabit fish instead of herbivorous animal Thus, the ova are developed only in fresh water, and for embryos, which are provided with six hooklets and numerous cilia; by means of the last they swim about freely. They are then swallowed by fishes, especially by pike and eel-pouts, and in their muscles the internal organs take on the form of cysticerci. The Bothriocephalus latus is found especially in Switzerland and Central Europe.

The **Symptoms** and **Treatment** are like those of the preceding. Recently some severe cases of anæmia have been traced to the presence of this worm.

ASCARIS LUMBRICOIDES.

Anatomy.—In shape and general appearance this resembles the ordinary garden worm (lumbricus); it is pink, cylindrical, and tapering at each end. The mouth is at one extremity, and is surrounded by three tubercles or lips provided with fine teeth; and it communicates with an intestine running the whole length of the animal. The female is from twelve to sixteen inches in length, and it has been estimated that the organs of generation can contain at one time sixty millions of ova. These measure $\frac{1}{340}$th of an inch in length, by $\frac{1}{440}$th in breadth. They have a dirty-brown colour, and are nodulated on the surface from the presence of an albuminous substance deposited outside the shell. They are found in the fæces of those troubled with the worm. There is still much doubt as to the manner in which the ascaris develops after the formation and escape of the embryo; but it seems probable that it requires an alternative host for its early stages in the same way as the tapeworms. The male ascaris is smaller than the female, and about ten inches in length; it is seldom met with.

These worms inhabit the small intestine, whence they may be passed *per anum*, or they may reach the stomach and be vomited. They have a curious tendency to insert themselves into apertures, or ring-like bodies, that may have been swallowed, such as the shanks of buttons; they have been found blocking the common bile-duct, the glottis, or the nasal passages; and occasionally they occur in abscesses in the groin or other part of the abdomen, about which it is not always easy to say whether the inflammation has been set up by the worm or by some other cause. The number of ascarides which may be present in the same individual is very variable; there may be only one, often there are only two or three, but sometimes they are in great number.

Symptoms.—These are not very different from those set up by tapeworms. On the other hand, there may be none. Nausea, foul breath, irregular appetite, itching of the nose, or abdominal pain may be present. In other cases there may be reflex symptoms, such as fits, choreic or convulsive movements, or mental disturbance. The migrations of the parasite lead to more serious troubles, such as jaundice, if it obstructs the bile-duct, or suffocation, if it gets into the larynx; and occasionally the worms have formed a convoluted mass large enough to cause intestinal obstruction.

Diagnosis.—Here, also, the diagnosis depends on the appearance of the worm or the discovery of its ova in the fæces. If a worm is discharged through the anus, or by vomiting, it is as well to treat the case as if others existed. Even when several have been expelled by treatment there may be others left behind; and this may be shown by the detection of the ova in the fæces. As specimens of the garden worm may be brought to the physician and passed off as ascarides, it should be remembered that the earthworm is redder in colour, less tapering at its extremities, provided with bristles along the sides to aid its progression, and that its mouth is a short transverse fissure on the under surface of the rounded head.

Treatment.—The best treatment for the Ascaris lumbricoides is the administration internally of santonica (wormseed), or its active principle, santonin. The latter is tasteless, and can be taken as a powder mixed with sugar, or simply placed on bread and butter, or suspended in milk. The dose for a child is 2 or 3 grains, for an adult from 4 to 6 grains; it should be taken on three or four successive mornings, and followed by a calomel purge, or a dose of compound rhubarb powder. Santonin sometimes produces inconvenient symptoms: the vision may be affected, so that objects appear green, yellow, or blue; or there may be tenesmus, or hæmorrhage from the bowels; and severe nervous symptoms, convulsions, and collapse have followed large doses. The urine is always coloured bright yellow, and is then turned red by the addition of an alkali.

OXYURIS VERMICULARIS.

The threadworm is very much smaller in size than the preceding. The female is about half an inch long, the male only one-sixth of an inch. They possess a mouth and complete alimentary canal, and the uterus of the female develops an enormous quantity of ova, which measure $\frac{1}{1100}$th by $\frac{1}{590}$th of an inch. The adult threadworms occur only in the large intestine, to a great extent in the rectum, where they are often matted together in balls; but also in the cæcum. There is, however, no new generation developed *in situ*, but the ova must be first taken into the stomach of the host, whether it be the same individual or another: and infection probably takes place from man to man, the ova drying on the clothes or on the skin and hair about the anus, and being conveyed by the fingers in scratching, or otherwise. And as the ova must also be present in the fæces of those affected, they may sometimes, from imperfect sanitary arrangements, get into drinking water, and be carried thereby to other people. The embryos are then set free in the upper part of the alimentary canal, and reach their full development in the cæcum, whence they generally move down into the rectum.

Symptoms.—These are mainly local, and due to the presence of the parasite in the rectum. The chief symptom is heat or itching at the anus, and this is worse at night, when the patient gets into bed, or shortly before this. There may be at the same time irritability of the bladder, with frequent micturition; or tenesmus, prolapsus ani, or excessive secretion of mucus; and in girls the worms may creep into the vagina, and set up irritation at the vulva, or vaginal discharge.

Treatment.—The use of purgatives, such as calomel, scammony, and jalap, will, of course, bring away some worms; but the object of treatment should be to kill them *in situ*. So far as the rectum and lower bowel are concerned, this may be effected by astringent enemata, such as infusion of quassia, with or without some solution of perchloride of iron, solution of alum (7 or 10 grains to the ounce), of tannin, of common salt, or of lime. The rectum should be cleared by a warm-water enema, and 5 or 6 ounces of the astringent should be injected, and kept in for some time. This should be repeated two or three times a week for two or three weeks. To destroy the worms resident in the cæcum it has been recommended to give saline purges frequently, or large doses of infusion of gentian or quassia internally. The itching at the anus is relieved by the application of unguentum hydrargyri.

TRICHOCEPHALUS DISPAR.

This nematode worm measures one and a half to two inches; in its anterior two-thirds it is extremely fine like a thread or hair, but the posterior third is thicker. It inhabits the cæcum, but rarely gives rise to clinical symptoms. The ova, which may be found in the fæces, have a long oval shape, and measure $\frac{1}{490}$th of an inch in length by $\frac{1}{1087}$th in breadth.

ANCHYLOSTOMUM DUODENALE.

This parasite, also known as *Sclerostomum duodenale*, and *Dochmius*, or *Strongylus duodenalis*, is a small nematode worm, which attaches itself in great numbers to the mucous membrane of the duodenum and jejunum. The female is a little more than half an inch in length, and the male about one-third of an inch.

The parasites live by sucking the blood of the intestinal mucous membrane into their own bodies, which thus acquire a pink colour. After the death of patients affected with this disease the worms are found in great numbers between the valvulæ conniventes, while the mucous membrane presents minute ecchymoses and blood is found extravasated into the cavity of the bowel. Those who are affected with these parasites suffer from a severe anæmia, which is explained by the extraction of blood from the intestinal vessels. There is a gradually increasing pallor of the face, lips, conjunctivæ, and body generally; puffiness of the face and feet; feebleness and lassitude, with quick, small pulse, palpitation, dyspnœa, and deranged digestion. The symptoms may go on for months or years, so that finally the sufferer dies from exhaustion, or from pneumonia. The disease occurs in many parts of the world, though not in England; the so-called "Egyptian chlorosis" is due to this parasite, and the numerous cases of anæmia occurring in 1880 among the workers in the St. Gothard tunnel, which were by some attributed to confinement underground, bad ventilation, &c., have been conclusively shown to be due to the Anchylostomum duodenale. It has been seen also in Brazil, and in Jamaica. The ova are probably taken into the stomach with impure drinking water.

The **Diagnosis** can scarcely be made from the anæmia alone, but from its occurrence in epidemic form the parasite may be suspected. Confirmation can be obtained by examining the fæces for ova, which are about the same size as those of the Oxyuris vermicularis; but the latter are more elongated, curved, provided with an operculum, and the embryo is already formed, while, in the case of the anchylostomum, the yelk is in the earliest stage of segmentation at the time that the ova are discharged.

Treatment.—Oil of male fern, santonin, and thymol have been used successfully in these cases, but the drugs require to be given in repeated doses, as the parasites exist in such numbers. Thymol may be given in cachet or wafer to the extent of 60 grains daily, in four to six doses. The anæmia is only slowly recovered from, and calls for the use of iron and general tonic treatment (*see* Anæmia).

TRICHINA SPIRALIS.

The disease known as *trichinosis* or *trichiniasis* is due to the nematode worm above named, which is found in enormous numbers in the voluntary muscles throughout the body, but which develops for the most part in the intestinal canal. The disease is rare in England, but is not uncommon in Germany. The intestinal worms are acquired by eating the flesh of pigs infested with the muscular trichinæ, and within forty-eight hours of its ingestion sexually mature intestinal trichinæ can be found. As usual, the females are more numerous and larger; they measure from one-twelfth to one-seventh of an inch, while the males are from one-twentieth to one-fourteenth of an inch, and differ from the females in presenting two small processes at the tail. Within seven days after ingestion, embryos are formed within the ova, and are discharged from the females already hatched. It would seem from recent observations that the females bore their way into the villi and other parts of the mucous membrane, and the embryos are deposited in the lymph or chyle vessels, by which they are carried ultimately to the voluntary muscles. In the muscles they increase in size, and possibly move about in the course of the muscular fibres. About the second week they reach the full size corresponding to this stage, namely $\frac{1}{25}$th of an inch, or a little less, and two or three weeks later they become coiled up, and, as a result of the irritation which they produce, are gradually surrounded with a capsule. This is oval, or rather fusiform with an oval bulging in the middle, and lies always parallel to the muscular fibres; it measures $\frac{1}{78}$th inch in length, by $\frac{1}{130}$th in breadth. It is at first nucleated and transparent, but afterwards becomes calcified, especially at its ends. Calcification in the human subject probably does not take place under twelve months, and even then does not interfere with the life of the parasite within. Indeed, it may remain in this condition for years; or it may perish, and be converted into a structureless mass. The muscles in which the trichinæ are deposited become of a pale reddish-grey colour, their fibres lose their striation, and become brittle and homogeneous, with numerous minute fissures. With the exception of the heart, all the striated muscles of the body may be affected, but the capsules are most abundant in the diaphragm, the intercostal muscles, the biceps, and the muscles of the larynx, and of the throat. As long as the worm remains alive in its capsule, it has the power of developing into a sexually mature trichina on being taken into the stomach of a suitable host.

Symptoms.—These consist mainly of febrile reaction with local evidences of inflammation of the muscles. In some cases there are at first gastro-intestinal disturbances, such as epigastric pressure, nausea, vomiting, and diarrhœa, or perhaps constipation. But these are often slight, and the commencement is, like that of many febrile diseases, characterised by loss of appetite, sleeplessness, lassitude, and depression. Very soon the arms and legs become painful; the knees and elbows are either flexed or extended, but in each case any alteration of the position is extremely painful, and the patient avoids every movement. The muscles of the limbs are tender, and feel hard and swollen to the touch. Mastication becomes painful, and the jaws may be closed for weeks; dyspnœa is produced by the implication of the respiratory muscles, and coughing, sneezing, and yawning are difficult or impossible. The inability to cough up the secretions aggravates the dyspnœa seriously. The movements of the eyeballs are painful, and the power of accommodation is said to be lost at the same time. Towards the end of the first week appears another symptom—namely, œdema. This occurs first in the eyelids, then the rest of the face and neck may be affected, and sometimes even the upper and lower extremities. Its causation is not clear. The fever is seldom very high, or continuous; the temperature is generally below 102°, but may rise to 104°. The pulse is rapid, there may be profuse sweating, and a miliary eruption; or in severe cases, vesicles, wheals, petechiæ, and pustules. The tongue is dry, red, and slightly furred; sometimes there are headache and stupor. Death takes place in the fourth or fifth week, or earlier, from exhaustion, pneumonia, or bronchitis.

Pathological Anatomy.—The only characteristic change is the condition of the muscles. There are sometimes signs of hæmorrhagic catarrhal inflammation of the small intestine; the liver is often fatty; the spleen is not enlarged.

Diagnosis.—There is a certain resemblance between trichiniasis and typhoid fever, in the febrile reaction and diffused pains, but with the progress of the disease the differences become marked. Cases of trichiniasis occur in groups, since an infected animal is likely to be eaten by many individuals or a family. A suspicion may be confirmed by the examination of the fæces or of a portion of muscle removed during life.

Treatment.—The embryos which have got into the tissues are beyond our reach; we can only hope to destroy the parasites in the intestine. For this purpose we may give castor oil or calomel in large doses. Benzine, 1 or 2 drachms daily, in gelatine capsules; glycerine, a tablespoonful every hour or two; and picric acid, ½ to 1 grain daily, have been recommended.

The muscular pains may be treated with narcotics internally, and chloroform or belladonna externally.

DISEASES OF THE LIVER.

The liver occupies the right hypochondriac region, under the ribs, and stretches across the upper part of the epigastrium. Normally it can scarcely be felt even in the latter situation, and there only when the abdominal parietes are very thin. Percussion gives dulness (*hepatic dulness*) in the mammary line, from the upper border of the sixth rib to the costal margin; in the middle line there is very slight loss of resonance for one and a half or two inches from the base of the ensiform appendix, where the thin left lobe lies over the stomach. When the abdominal parietes are thin the edge of the liver may be perceptible to sight during deep inspiration, as the organ descends for one and a half inches, and the percussion dulness shifts to a corresponding extent. In the axillary line the hepatic dulness begins at the eighth rib, and at the tenth rib in the line of the scapular angle.

In disease the organ is often enlarged. It then, as a rule, projects below the costal margin, and can be felt more or less readily. It may reach to the level of the umbilicus or much lower, its lower margin extending across the abdomen from the right flank to the left costal margin. It presents different degrees of consistence, and alterations of surface, according to the disease affecting it. The liver only encroaches on the chest when the enlargement is of a local kind, such as cancer, hydatid, or abscess, or when it is itself pushed up by something below.

Apparent enlargement of the liver arises from tight lacing, and from tumours or pleuritic fluid in the chest. The former elongates the organ vertically, squeezing out, as it were, a portion below the ribs; the organ has its natural consistence, and the cause is usually obvious. In the latter the whole liver is displaced.

In atrophy of the liver, there is a diminution of the percussion dulness, but a similar diminution may be caused by the encroachment of intestines distended with air.

A distended gall bladder may be perceived as a globular prominence at the lower border of hepatic dulness, in the mammary line.

Two common results of hepatic disease are jaundice and ascites, and these conditions will be discussed before the special diseases of the liver are described.

JAUNDICE.

By the term jaundice (from *jaune*, yellow), or *icterus*, is meant a yellow discoloration of the skin and other parts by bile-pigment circulating in the blood. In ordinary cases the skin has a more or less deep yellow tinge, the conjunctivæ are yellow, and the visible mucous membranes have their natural red colour obviously modified by the yellow tint. In long-standing cases the colour of the skin becomes deeper, and finally of a greenish or olive-brown tint. This, formerly distinguished by the name of *black jaundice*, is due, no doubt, to the gradual conversion by oxidation in the skin of bilirubin, the yellow pigment of the bile, into biliverdin. The yellow colour must be distinguished from other changes of colour in disease, such as the yellowish tinge of cases of chlorosis and of pernicious anæmia, the sallow tint of malarious cachexia, and the brown colour of Addison's disease. The colour can be generally well recognised in the conjunctiva, but in some people small masses of subconjunctival fat give a tint which is not very unlike it.

The colour of the *urine* is at the same time altered, from the presence of the biliary pigment. In small quantity this gives it a bright saffron colour, which is best seen in any froth which may form on the surface; if there is more, the urine becomes brownish-yellow, or yellowish-brown, or even dark brown like porter. If linen or paper is dipped in the urine, it is stained bright yellow; but the presence of bile-pigment can be more certainly proved by the application of chemical tests which will be mentioned presently. Of the other secretions of the body the majority are not discoloured: occasionally the sweat is tinged yellow, and sometimes the milk of nursing women. The tears, saliva, gastric juice, and intestinal secretions are unaffected; but the secretions from the mucous membranes, when diseased, and more frequently morbid effusions from serous membranes, may contain some biliary pigment.

In most cases of jaundice the *fæces* are altered in colour, becoming whitish or clay-coloured; this is due to the absence from them of a derivative of the bile-pigment, stercobilin, in those cases of jaundice where the bile is unable, from obstruction of the ducts, to find its way into the duodenum. The absence of bile from the stools may have other effects, since this secretion is known to have some share in the digestion of fat, and is thought to have a power of preventing putrefaction in the intestinal contents, and to stimulate the muscular fibres of the intestinal wall. Accordingly, it is common to find fat in the stools; they are sometimes offensive, and constipation is frequent, though by no means invariable. When diarrhœa occurs, it has been attributed to the irritation of the putrescent fæces.

Other symptoms are often present in jaundice, which are no doubt due to the circulation in the blood of the constituents of the bile. Occasionally the *pulse* becomes slowed to fifty or forty per minute. This is probably owing to the action of the bile-acids upon the cardiac ganglia. It is most common in cases of catarrhal jaundice. *Itching* occurs, especially when the jaundice is due to obstruction of the bile-ducts; and it may be so intense that sleep is rendered impossible, and blood-crusts, papules, or wheals of urticaria are produced by the incessant scratching. The cause of the itching is uncertain - it is at least not due to the bile-pigment; indeed, it has been noticed in cases some time before the jaundice appeared. A curious disease of the skin named *Xanthelasma* or *Xanthoma* occurs in some cases of chronic jaundice after many months (*see* Diseases of the Skin).

Some patients have a bitter taste in the mouth, and digestive disturbances are frequent. In certain forms of jaundice hæmorrhages take place under the skin or from the mucous surfaces, and serious cerebral symptoms arise, such as delirium, convulsions, and coma; but these, again, are not the result merely of the circulation of bile-pigment in the blood.

Tests for Bile-Pigment in the Urine.—The essential feature of these tests is the production of a green colour by the conversion (by oxidation) of yellow bilirubin into green biliverdin: in some processes, other tints are temporarily developed. The most commonly employed is Gmelin's. A few drops of urine are placed upon a white plate, and a little strong nitric acid is dropped close by, and then the two fluids are gently run into one another. At the line of contact, the colour of the urine changes, becoming green, blue, violet, red, and lastly yellowish or brown. The urine may be slowly poured by means of a pipette on to the surface of nitric acid, placed in a test tube, when a similar result will be obtained. If nitric acid be added to the diluted urine in the test tube, the mixture will turn green.

The oxidation may be effected by iodine: if a few drops of a solution be dropped on to the surface of the urine in a test tube, the urine becomes of a deep green colour (Maréchal's test).

Tests for Bile-Acids in the Urine.—For Pettenkofer's test, Halliburton directs that 100 to 200 c.c. of urine shall be evaporated to dryness on a water-bath, and the residue extracted with absolute alcohol; the extract is filtered, and twelve or twenty times its bulk of ether is added. This precipitates the bile-salts, which are collected, dissolved in water, and decolorised with animal charcoal. The liquid may then be tested by adding a strong solution of cane sugar to it in a capsule: to this a drop of sulphuric acid is added, and the whole gently warmed. If bile-salts are present, a purple colour, which is due to a combination of purpuraldehyde with cholic acid, appears.

Oliver uses peptones as a test. The urine is filtered, acidified if necessary, and diluted to a specific gravity below 1008. Twenty minims are then added to 60 minims of the following solution:—Powdered peptone (Savory & Moore's), ʒss.; salicylic acid, 4 grains; acetic acid, ʒss.; distilled water to 8 ounces, filtered till quite transparent. If bile-acids are present, milkiness appears at once; it may disappear on agitation, but reappears on adding more of the test.

Theories of Jaundice.—It is not difficult to explain the occurrence of jaundice in the cases in which the outflow of the bile is prevented by any stricture or obstruction of the common bile-duct. The bile distends the gall-bladder and the bile-ducts, and then passes into the lymphatics and blood-vessels, circulates in the latter, and gives the characteristic tinge to the skin and other parts. An interesting fact in the secretion of the bile makes it likely that a complete obstruction is not necessary—that is, that the bile is secreted under very low pressure, such that in guinea-pigs a pressure of twenty centimetres of water will force the secreted bile back into the circulation. Where there is complete obstruction, as from a gall-stone in the common duct, or a cancerous tumour pressing upon it, the bile is unable to reach the intestines, and the fæces, as already stated, are white or clay-coloured. This is called *obstructive* jaundice. If the obstruction is removed—as, for instance, by the gall-stone passing at length into the duodenum—bile again flows into the intestine, the fæces becomes dark, the urine acquires a normal colour, and the skin more slowly loses its jaundiced hue.

It is not so easy to account for cases of jaundice in which the bile-ducts are not obviously obstructed; and it is in explanation of this difficulty that so many theories have been brought forward. It has been held that the jaundice was due to *suppression* of the bile-secreting function of the liver, bile-pigment being formed in the blood, and passing into the urine and tissues, because it could not get away through the liver. Another view is that the jaundice is due to excessive destruction of hæmoglobin, producing pigments beyond what even the healthy liver could secrete. Murchison's view was that the normally secreted bile might be produced and re-absorbed from the bowel in such quantity as to be incapable of destruction by the respiratory processes. None of these theories are adequate; and the explanation probably lies in certain changes which the bile undergoes in cases of non-obstructive jaundice, so that its absorption into the lymphatics becomes necessary.

If a particular organic substance named *toluylendiamine* is taken internally, it causes jaundice; and this it appears to do by destroying the blood-corpuscles, and liberating hæmoglobin. The hæmoglobin being carried to the liver increases the quantity of bilirubin, and the bile thereupon secreted, contains much bile-pigment, but little bile-acid. Though at first secreted in quantity, it soon becomes thick, viscid, and tenacious, and so increases the pressure in the bile-ducts, that the secreted bile is absorbed by the lymphatics and carried into the circulation, and jaundice results. W. Hunter believes that this viscidity of the bile is brought about by an intense catarrh of the minute bile-ducts, the result of irritation by the poison itself, or its products in the bile.

Some other forms of jaundice, such as those produced by phosphorus, arsenic, and antimony, and the jaundice which occurs in association with numerous infectious diseases, enteric fever, relapsing fever, yellow fever and others are probably to be explained in the same way. Thus, nearly all forms of jaundice arise by absorption of bile secreted in the liver and delayed in the ducts either by mechanical obstruction of the hepatic or common bile-duct, or by actual or relative obstruction in a number of small ducts. The actual obstruction is narrowing of their channels—the virtual obstruction, such an increase or alteration of the secretion as to make it flow too slowly through them, or such alteration in the relative pressure of bile in the small ducts and fluid in the vascular system, as will drive the bile-pigment into the latter.

Causes of Jaundice.—These are:—

Obstruction of the Larger Bile-ducts:—(1) Gall-stones and inspissated bile, hydatids, distomata, foreign bodies from the intestinal canal; catarrhal or inflammatory swelling of the wall of the bile-duct. (2) Stricture or obliteration of the duct from congenital defect, from perihepatitis, or from former ulceration of the duodenum or of the bile-duct itself. (3) Compression by tumours of the liver, stomach, pancreas, kidneys, omentum, ovaries, or uterus, by an abdominal aneurysm, accumulated fæces, or pregnant uterus.

Probable Obstruction of the Minute Ducts:—(1) By tissue changes; cirrhosis and different forms of chronic atrophy. (2) By increased viscidity of the bile in the minute bile-ducts, the result of catarrh; poisons such as phosphorus, arsenic, antimony, mercury, and others; snake poison; infectious diseases, namely, typhus, enteric fever, relapsing fever, yellow fever, malarial fevers, scarlatina, pyæmia, and possibly acute pneumonia; different forms of acute, febrile, malignant, and infectious jaundice, acute yellow atrophy, and Weil's disease; acute and chronic congestion of the liver, probably by means of catarrh of the ducts.

The discrimination of the various forms of jaundice must depend on a consideration of the diseases to be presently described; but it may here be pointed out that the most common forms in English practice are catarrhal jaundice, and those associated with gall-stones, with cancer of the liver and bile-ducts, and with cirrhosis.

ICTERUS NEONATORUM.

Jaundice in infants is sometimes due to congenital obstruction of the duct. More often it is a merely temporary condition which passes off in a few days, and is probably due to retention of bile in the small ducts; but the cause of this is by no means clear.

ASCITES.

By this term is meant the presence of serous fluid in the peritoneal cavity. Like other effusions into the serous cavities, it is commonly alkaline, of a pale straw colour, of specific gravity 1015 to 1018, highly albuminous, and containing chlorides. It arises (1) whenever the portal circulation is obstructed, either in the trunk of the portal vein, or in its distribution in the liver; (2) as a result of diseases of the peritoneum; and (3) as a part of the general dropsy of renal disease.

The portal vein trunk may be obstructed by the pressure of tumours, and enlarged glands in the portal fissure, by cancer, abscess, or hydatid in the liver itself, and by coagulation of blood in its interior (*thrombosis*, *pylephlebitis*). In the liver the chief cause of portal obstruction is the compression of the interlobular veins by the fibrous overgrowth of cirrhosis; another cause is perihepatitis; a third kind of obstruction is formed by the varieties of cardiac and lung disease, in which the right side of the heart is dilated, and the passage of the blood through the chest is impeded (*see* Diseases of the Valves, and Emphysema of the Lungs).

The peritoneal diseases causing ascites are—acute and chronic peritonitis, tubercular peritonitis, and cancer of the peritoneum.

In Bright's disease, the peritoneum is the seat of effusion in common with the other serous cavities.

The **Physical Signs** of ascites must be carefully attended to, as it is not impossible to confound it with other conditions. The abdomen of course enlarges, and in the early stages of a considerable ascites, it is generally tense, and the form tends to be globular, with a decided prominence in a forward direction. Later the walls of the abdomen become stretched, and as the patient lies in bed the fluid gravitates backwards, and gives a broader and flatter shape to the belly. The liquid then poured out may amount to three, four, or five gallons, and the abdomen becomes proportionately enlarged so that it may measure from forty to forty-two inches or more in circumference. The presence of fluid is detected by three methods of examination—*percussion*, *fluctuation*, and *displacement*.

Percussion.—Normally, the surface of the abdomen is resonant, from the air contained in the stomach and intestines; but when fluid is poured out, this collects at first in the flanks and hypogastric region, so as to give a dull note to percussion in these parts, while the centre of the abdomen remains resonant.

As the fluid increases, the dulness encroaches more and more from the sides and hypogastrium upon the centre, and at length only a limited area remains resonant—namely, that which includes the umbilical and the left hypochondriac regions. If, in either of these stages, the patient be turned upon one side and again percussed, it will be found that the anterior and central regions have become dull, and the flank, which is now uppermost, gives a resonant note. This is due to the gravitation of the fluid to the lowest part, and the floating of the air-containing bowel to the highest; and this occurrence is the most conclusive proof of the presence of fluid in the peritoneum. Occasionally, however, the abdomen is entirely dull, where the mesentery is so short, or the fluid so abundant, or the viscera are matted down by chronic peritonitis in such a way, that the intestines cannot float to the uppermost part. Then also this test by change of position fails to give the desired information.

Fluctuation is obtained by laying one hand on one side of the abdomen, and sharply tapping or flipping the other side with the finger. The applied hand then feels the transmission of a wave across the abdomen. This is a less certain sign than the former. Very fat abdominal walls may transmit a wave without the presence of fluid, and to prevent this, the edge of the hand, or of a book or card, should be pressed on the centre of the abdomen while fluctuation is tried.

The method of *displacement* has only a limited application. If in a case of ascites the liver is enlarged, it sinks in the fluid, and a small quantity of fluid lies between its anterior surface and the abdominal wall. By placing the fingers on the abdomen at this spot, and suddenly pressing them in, the fluid is displaced, and the surface of the liver may be felt. This method, which enables the liver to be recognised, is at the same time proof of the presence of fluid; since, if there were no fluid the liver would be in close apposition with the anterior abdominal wall.

Ascites is, however, sometimes simulated by one or other of the different kinds of cysts which may occur in connection with the abdominal or pelvic viscera, by a pregnant uterus, or by a distended urinary bladder. These cysts are ovarian, hydatid, or renal cysts. They are excluded if the percussion test is successful; on the other hand, they may give the fluctuation test; and if the whole surface is dull there may be some difficulty in distinguishing between one of those and an ascites in which the intestines are bound down. An operation for ovariotomy has several times been attempted, at which the case has been proved to be one of ascites. Ovarian dropsy is chiefly distinguished by the abdomen being dull in front and resonant in the flanks, into which position the intestines are pressed by the cyst; and by the swelling at least beginning on one side, though it is afterwards central. Not infrequently also the outline of the cyst can be recognised at the uppermost part, especially if looked for during the movements of respiration. Some of the measurements of the abdomen are different in the two cases; thus, normally, and in ascites, the umbilicus is about an inch nearer to the pubes than to the sternum; in ovarian cysts this ratio is often reversed. In the latter, also, the distance from the umbilicus to the crest of the ilium may be greater on the side of the diseased ovary, and the greatest girth of the abdomen is an inch or two below the umbilicus; whereas in ascites it is at the umbilicus or a little above it. Lastly, the fluid withdrawn by tapping an ovarian cyst is often grumous, brown or chocolate-coloured from containing altered blood.

Rarely it happens that chyle is extravasated from ruptured lacteal vessels into the peritoneal cavity, constituting what is known as *chylous ascites.*

CIRCULATORY CHANGES IN THE LIVER.

ANÆMIA.

This does not occur as a separate affection. The liver suffers with other organs in conditions of general anæmia; fatty, lardaceous, and cirrhotic livers doubtless contain less blood than normal; and lastly, after death it is not uncommon to find patches of a pale colour due to local anæmia, but they have no clinical significance, and are possibly, indeed, of *post-mortem* occurrence.

PASSIVE CONGESTION.

The *nutmeg liver*, which has been already described (p. 512) as one of the results of valvular disease of the heart, is an extreme form of passive congestion. In earlier stages of the same change the organ is simply engorged, being larger than normal, and dark red in colour, with obvious distension of the intra-lobular veins. It causes fulness and discomfort in the right hypochondrium, and ultimately ascites and slight jaundice. Hepatic pulsation is occasionally ascociated with it.

ACTIVE CONGESTION.

Active congestion of the liver no doubt arises in the course of various febrile illnesses, and it must form a part of acute inflammation of the organ. It is common also to regard as due to active congestion the symptoms which occur in those who have attained thirty-five or forty years of age, who live sedentary lives, and eat and drink freely. These are a sense of weight, oppression, or fulness in the right hypochondrium, increased by pressure, by tight clothes, or by lying on the left side; pain in the right shoulder, furred tongue, nausea or sickness; slight jaundice, constipation, and the passage of high-coloured, scanty urine, depositing urates. In addition the patient suffers from a bitter taste in the mouth; aching pains or severe cramps in the limbs; lassitude, drowsiness, headache, and giddiness; grinding of the teeth; sleeplessness; palpitation, fluttering, and intermittent action of the heart. The congestion of the liver is no doubt set up by the conditions of gastric or intestinal catarrh which occurs in such patients. Some of the symptoms are directly due to the same causes. Probably some toxic agents are in operation, especially when one remembers the frequency of hepatic congestion verging on inflammation, which occurs in the tropics, both in connection with excesses, and with malaria and dysentery.

Treatment.—This consists in the careful regulation of the diet, taking active exercise, and the use of mercurial and other purges. Alcohol should be limited to a little claret or Rhine wine with dinner; and meat, spiced and made dishes, should be taken only in small quantities. When the patient is actually suffering, the best remedy is pil. hydrargyri (2 to 4 grains) with pil. coloc. co. (3 to 4 grains), followed by mist. sennæ co. or sulphate of soda or magnesia. The bowels may further be kept active by occasional saline purges, or the daily use of Püllna, Friedrichshall, Carlsbad, or Hunyàdi János waters.

ACUTE HEPATITIS.

Acute inflammation of the liver is not a common affection in England, and in tropical countries it often ends in abscess. But it may terminate by resolution in a week or ten days. Its symptoms are essentially the same as those which precede the development of suppuration (*see* Abscess of the Liver).

ABSCESS OF THE LIVER.

Pathology.—Abscesses of the liver arise under a variety of conditions, but what is common to nearly all of them, except injury, is the introduction of some septic agent by one of three channels: the hepatic artery, the portal vein, or the bile-ducts. In the first case they form part of a general pyæmia, such as results from wound or injury in any part of the body, but especially injuries to the head; they are small in size, and numerous, or at least multiple.

They are known as *pyæmic abscesses*. The portal vein is responsible for a still larger number of cases, the septic agents being carried from lesions within the portal vein area, such as gastric ulcer, appendicitis, pelvic suppurations, and especially ulcerative lesions of the intestine, including tropical dysentery. Under these different circumstances the abscesses may be single, few, or multiple; and in the last case especially the condition may be spoken of as *portal pyæmia*. Sometimes the portal vein and its branches are filled with broken-down purulent clot, and the walls of the veins are inflamed, constituting *suppurative pylephlebitis*. From this group of cases it is difficult to exclude the well-known *tropical abscess*, which is constantly seen in association with dysentery, but which is believed by some observers to arise independently. In support of this last view it is stated that cases of hepatic abscess occur without any history or symptoms, or *post-mortem* evidence of dysentery, and that the symptoms of dysentery sometimes follow hepatic abscess instead of preceding it. The frequency with which tropical abscess is solitary and of large size lends colour to the view that it is different from the multiple small abscesses of many cases of portal pyæmia. But multiple abscesses sometimes occur in dysentery; and out of the tropics large single abscesses sometimes follow ulcerative lesions of the intestine which are not dysenteric. Moreover, the discovery of the *amœba coli* in abscesses of the liver associated with dysentery due to the same organism, shows that in some cases at least there must be a causative connection between the two lesions. Invasion by the bile-ducts is chiefly effected in consequence of gall-stones ulcerating into them.

The difference to be noticed in the symptoms in various cases of hepatic abscess makes it desirable to consider separately the multiple abscesses, which are generally pyæmic, and the large solitary abscess, which is so often tropical.

MULTIPLE (OFTEN PYÆMIC) ABSCESSES.

The abscesses vary in size from a pin's head up to that of a hazel-nut; they may contain well-formed pus, or sanious fluid and *débris*, or more bulky sloughs that have only just been separated. In cases originating in pylephlebitis it may be easy to show that much of the suppuration is in the course of the distribution of the portal vein. The capsule of the liver is frequently inflamed where abscesses approach the surface. The abscesses found in the liver by Councilman and Lafleur in connection with amœbic dysentery (p. 101) were single or multiple and presented some peculiarities. They always contained amœbæ, and never any so-called pus-organisms. The process was not a true suppuration, but a softening and liquefaction of tissue, already largely necrotic.

Symptoms.—Cases of multiple abscess in the liver are often very obscure, especially when they form a part of a general illness like pyæmia. There is severe constitutional disturbance, with fever of hectic type, rapid pulse, dry brown or furred tongue, and early prostration. Vomiting is often present, but the action of the bowels is variable; sometimes there is constipation, at others diarrhœa. The liver is mostly enlarged, and in some cases may reach to the level of the umbilicus; it is painful and tender. Jaundice is sometimes, but not necessarily, present; it probably requires the compression by an abscess, or the obstruction by gall-stones, of some larger bile-duct. The condition of the urine and of the fæces as to the bile-pigment will, of course, vary with it. The duration of the illness is from one to several weeks, but the end is certainly fatal.

Diagnosis.—This must depend on the fact that the liver is involved in an acute process, with severe constitutional disturbance, especially if it is associated with some lesion which can be recognised as the primary cause. The presence of jaundice very much facilitates the diagnosis, and Fagge distinguished such cases as *febrile jaundice.* Where jaundice is absent, it may have to be distinguished from tropical abscess by the uniform enlargement of the liver.

The **Treatment** must be mainly symptomatic. An attempt must be made to improve the general condition by nourishment, quinine, and stimulants. Opium and local applications, poultices, fomentations, &c., will be required to relieve pain.

Large, Solitary (often Tropical) Abscess.

This is met with frequently in India and other tropical countries. It occurs especially between the ages of twenty and forty-five, the intemperate are more liable to it than others, and it has a relation to dysentery, which has been already discussed.

Anatomy.—Tropical abscess is usually, though not always, single, and may reach a considerable size. Its condition varies somewhat with its age: thus a recent abscess presents a ragged inner surface, with little that can be called lining membrane; a somewhat older cyst is lined with opaque yellowish deposit; while very old abscesses are surrounded by a dense fibrous wall formed in the liver-tissue surrounding them. The pus contained in them is either whitish or yellow like pus from other sources; or greenish in colour, and viscid, or curdy; or it may be red or reddish-brown from mixture with blood. It has a peculiar nauseous odour, but is rarely offensive unless the abscess is near the colon. The characters of the amœbic abscess have been described. In abscesses free from amœbæ, staphylococci (aureus and albus) have sometimes been found. The amount of pus may reach five or six pints or even more.

It is probable that the pus from small abscesses may be absorbed, and in some cases contracted cavities with cretaceous remnants have been found in the liver, indicating a former abscess. But, as a rule, the abscess after a time makes its way to the surface of the liver, where it sets up perihepatitis, and ultimately opens either externally through the abdominal parietes, or into the stomach, duodenum, or colon, or into the abdominal cavity, or by perforating the diaphragm into the pleural cavity or lung. Sometimes it opens externally through an intercostal space, having first perforated the diaphragm and displaced the lung. It may thus be rapidly fatal, causing peritonitis, pleurisy, or pneumonia; or the cavity may contract after free discharge externally, or into the lung or into the bowel.

Symptoms.—In the early stages these consist of chilliness or actual rigor, followed by febrile reaction. Locally there is pain in the right hypochondrium, which may reach up to the right shoulder; and fulness and tenderness in the same region, with some evidence of enlargement of the organ. There is frequently, but not always, some jaundice. The movement of the ribs in respiration is impeded, and there is a short, dry cough. In the course of a week or a fortnight there may be evidence of suppuration. The liver is enlarged, and may reach far down into the abdomen; the enlargement, however, is not uniform, but there is a marked prominence at one or other part. If there is a large abscess in the right lobe, the ribs of the right side may be elevated so as to cause an obvious bulging of the lower part of the chest, and to increase on that side the costal angle—*i.e.*, the angle between the margin of the ribs and the middle line. An abscess projecting from the convex surface of the liver at its back part will cause dulness at the base of the chest by displacement or compression of the lung, diminishing at the same time the breath-sounds, so that an empyema is simulated. If the abscess bulges in front, the surface is smooth, elastic and tense; and fluctuation may be felt, although it is often absent if the abscess is deep-seated and surrounded by much thickness of hepatic tissue.

The pain is at first dull and heavy, becoming more severe as the abscess reaches the surface, and either sets up peritonitis or stretches the integuments. Pain is felt, also, when the patient lies on the left side, from the falling of the liver towards that side. Both pain and tenderness may, however, be absent. Ascites and marked jaundice are not commonly present; when they are, they result from direct compression of the portal vein or of the large bile-ducts. With the formation of the abscess, the constitutional symptoms become pronounced. The fever is irregular, and of hectic type. Occasionally there are rigors, during which the temperature rises to 103°–105°, falling with profuse perspiration. The tongue is furred or dry, and vomiting may be frequent, but the bowels are variable.

In course of time there is severe prostration, with emaciation, and the skin assumes the sallow tint common with extensive suppuration. Subsequent symptoms depend on the course which the abscess takes; if it points externally, there is increased prominence at the spot selected, the skin becomes red, tender, and œdematous; its invasion of the chest may cause for some little time the symptoms of compression of the lung above indicated, and finally the chest-wall may bulge opposite one of the intercostal spaces. If it points more centrally, pleurisy, serous or purulent effusion, or pneumonia may occur; or the lung may become adherent to the diaphragm, so that the abscess opens directly into the bronchial tubes, and the purulent contents are gradually expectorated. Pericarditis, from rupture of the abscess into the pericardium, is generally quickly fatal. It is important to remember that sometimes the symptoms of hepatic abscess are entirely latent until one of these accidents by rupture occurs.

The duration of abscess of the liver is variable; it may be from a few weeks to three or more years.

Diagnosis.—The diseases which may be confounded with abscess of the liver are acute perihepatitis, suppurating hydatid (which is, indeed, an abscess of the liver arising in another way), suppurating gall-bladder, subphrenic abscess, empyema, abscess of the abdominal wall and pyelitis. Hepatic abscess, otherwise simulating empyema, often differs from it in the upper line of dulness being convex, and falling near the spine, instead of directly transverse. The fever of hepatic abscess is often mistaken for ague, but will not yield to quinine, as does the latter.

Prognosis.—Some cases get well under treatment, and occasionally this happens when the abscess has discharged into the lung or bowel; but a fatal result is very frequent. On the whole, the smaller abscesses may be considered more favourable.

Treatment.—In the earlier stages, writers recommend salines, chloride of ammonium, and ipecacuanha: the latter should be given in doses of 18 to 20 grains, repeated every six or eight hours, and is believed to be as valuable in this disease as in dysentery. When suppuration has occurred, pus should be evacuated, if possible. Until this operation can be undertaken, the patient should be supported by nourishing diet, and the moderate use of stimulants; and pain should be relieved by opium and morphia, and the application of fomentations and poultices. The evacuation of the pus forms a surgical operation:

it may be done by the aspirator, the use of which alone is sometimes enough to effect a cure; but where the abscess can be reached by an incision, a free opening and drainage are preferable.

ACUTE YELLOW ATROPHY.

In this remarkable disease the liver undergoes a rapid degeneration of its tissue, and diminishes in size to two-thirds, or even one-half, of its normal bulk.

Ætiology.—It is more common in females than in males, and the majority of patients are under thirty years of age, though it is very rare in children. Indeed, at any age it is a disease of extreme rarity: the records of a large hospital will not show one case in a year, and it must fall to many medical men never to have seen an instance of it. As to its cause, it is certain that its onset is often preceded by severe mental emotions, and many of the cases have occurred in people who have led a dissipated life, in women of loose habits, in the subjects of constitutional syphilis, and in women who are pregnant.

Symptoms.—The symptoms are at first obscure. Often it begins with a jaundice indistinguishable from catarrhal jaundice; or with gastro-intestinal symptoms, such as nausea, vomiting, and irregularity of the bowels; and pains in the hepatic region may occur comparatively early. These symptoms may last two or three weeks, when the more characteristic features develop. These consist of marked cerebral disturbances—at first headache and restlessness, then delirium and gradually developing coma, with convulsive twitchings, or more rarely epileptiform fits, towards the end. Jaundice then appears, or if it have been present early, it becomes deeper. The temperature is rarely high, but may be from 101° to 102°. The pulse, which may have been slow with the early jaundice, now becomes quick. The tongue is dry and brown, and as the symptoms progress, sordes collect about the lips and teeth. There is, besides, pain in the hepatic region, and decided tenderness, which may be recognised even during the stage of coma, if pressure be made there. The extent of dulness diminishes with great rapidity, so that, finally, it only measures, vertically, an inch or less.

The abdomen is natural, or towards the end it is retracted. The spleen is mostly enlarged. The urine contains bile-pigment, but generally in small quantity; it is said that the bile-acids have been found. But the most remarkable change in the urine is the extraordinary decrease in the amount of urea, uric acid, and salts (chlorides, phosphates, and sulphates), and the presence of two new compounds—leucin and tyrosin. The urea may be entirely absent. Leucin and tyrosin can be obtained by simply evaporating a small quantity of urine on a glass slide or in a watch glass, when their crystalline forms may be recognised under the microscope. Leucin forms circular plates, with concentric markings upon them; tyrosin crystallises in fine needles, which are arranged in sheaf-like bundles or in globular masses. These bodies hold chemically a position intermediate between albumen on the one hand, and urea and uric acid on the other, and their presence seems to be explained by the destruction of the liver, which is now unable to carry out its urea-forming functions. The urine not infrequently contains albumen, especially towards the end; and there may be also blood. This appearance of blood in the urine is part of a general hæmorrhagic tendency. There may be coffee-ground vomit; the fæces, which appear to be mostly pale, and deficient in bile, often contain blood; and there may be epistaxis, metrorrhagia, or petechial hæmorrhages under the skin. With increasing coma, death finally takes place, the severer symptoms lasting only from two to four days. Pregnant women, as a rule, abort.

Anatomical Appearances.—The liver is very much diminished in size; it may be only thirty-one or twenty-eight ounces in weight. It is soft, flaccid, almost like a bag of fluid, and its capsule, which is wrinkled, appears too large for its contents. On section, the liver is of a yellow colour, with patches of rather bright red; or in some parts it is entirely red, in others all yellow. The essential change is a granular and fatty degeneration, by which the hepatic cells are more or less completely destroyed. In the yellow parts of the liver the destruction is less advanced, and some bile-stained cells may still perhaps be found. In the red parts the colour is due to the more complete degeneration of the tissue, by which the vessels are left alone to represent the substance of the liver. Under the microscope, one can often see nothing but granules of albuminous matter, fat and pigment, and larger globules of fat. Leucin and tyrosin are also found in the liver, and will spontaneously crystallise on the surface of sections some hours after death. The bile-ducts are empty, and not stained by bile-pigment; the gall-bladder is also empty, or contains a small quantity of viscid grey mucus.

Other organs undergo fatty degeneration, especially the kidneys, in which the secreting cells are granular and fatty, and the heart and muscles. Petechiæ are found not only under the skin, but in the mucous membranes, under the serous membranes, in the kidneys, and other parts. The blood is thin and fluid, and leucin and tyrosin have been found in it.

Pathology.—As to the nature of the disease, different views have been held. Bright described a case as subacute inflammation, and its inflammatory nature has been accepted by others.

There is much in favour of its being an infectious disease—namely, the petechiæ, the splenic enlargement, the condition of the blood, and the typhoid, or adynamic condition preceding death; but micro-organisms have only been found in a few cases. The jaundice of acute yellow atrophy is explained by acute catarrhal changes in the minute bile-ducts.

Diagnosis.—This can only be made when the cerebral symptoms commence; it is then generally easy. Occasionally, however, jaundice, an important guide to diagnosis, has been absent. The disease is closely simulated by phosphorus-poisoning, in which the liver undergoes fatty degeneration, and there are jaundice, petechiæ, and cerebral symptoms. But in phosphorus-poisoning the liver is very much enlarged at first if not throughout, and leucin and tyrosin are less abundant in the urine, if present.

Prognosis.—Acute yellow atrophy is almost invariably fatal. A few cases are believed to have recovered; in one such case which occurred at Guy's Hospital, the patient died of the same disease a few months later.

Treatment.—This must, at present, be purely symptomatic. Sickness, constipation, headache, hepatic pain, and later the active delirium or retention of urine, are the conditions that may have to be met.

CIRRHOSIS OF THE LIVER.

(*Chronic Interstitial Hepatitis.*)

This is a chronic inflammation of the liver, which results in an extensive growth, usually in the course of the portal canals, of a contractile fibrous tissue, whereby the secreting cells of the liver are compressed and destroyed, while the course of the blood through the portal system of veins is seriously obstructed. The liver in advanced cases presents, on section, a number of yellow, brownish-yellow, or brown lobules of hepatic tissue, surrounded and separated from one another by broad tracts of grey translucent fibrous tissue; and it was on account of this generally yellow colour that the term cirrhosis (κιῤῥός, yellow) was used, and not in reference to the presence of excess of fibrous tissue. Nevertheless, the name has been often applied to chronic fibrous changes in other organs of the body—*e.g.*, cirrhosis of the lung and cirrhosis of the kidney.

Ætiology.—In the great majority of cases the cause of cirrhosis is the excessive use of alcohol, in the form of beer, wine, or spirits. Little is known as to the amount that is required to produce cirrhosis; there are the widest individual differences. Some people may drink freely all their lives without acquiring it, whereas in others a few months' indulgence seems sufficient for the purpose. In some children that have been the subjects of it, the fact of alcoholism has been proved.

But the simple theory of direct irritation by alcohol has not escaped criticism, and the following views have also been advanced: That the real irritant is some toxin produced in the mucus which results from the accompanying gastritis: that the irritant is not alcohol, but some other constituent of the liquid drunk. The excessive use of spices is said to be occasionally the cause. It is also said that infectious diseases, such as scarlet fever, measles, or pneumonia supply the requisite toxin for the occurrence of the common forms of cirrhosis. Somewhat different forms of cirrhosis are due to malaria and congenital syphilis; and much less pronounced degrees of fibrous overgrowth appear to result sometimes from heart disease, rickets, and very rarely tuberculosis.

The growth of connective tissue which has been found to follow ligature of the common bile-duct in animals, is not represented by any similar change in man.

Anatomical Changes.—The cirrhotic liver varies considerably in size. It may be so large as to reach during life two inches below the level of the umbilicus, and to weigh after death as much as eight or ten pounds; it may be so small as to be inaccessible to touch, and to weigh only twenty-eight or thirty ounces; and it may be of any intermediate size. The larger livers are often smooth, or only slightly granular on the surface; the smaller livers are coarsely granular, or nodular, or present large round bosses, or are distorted into curious shapes. In all cases the organ is very much tougher and harder than normal, from the development of fibrous tissue, which runs in all directions through it. If it can be examined in the earliest stages of cirrhosis, there are found large numbers of leucocytes infiltrating the tissue about the portal canals (Glisson's capsule), and in some cases penetrating more or less between the lobules, or even within them. From these leucocytes is developed white fibrous tissue, which forms a large part of the section in an advanced case. The bands of fibrous tissue running through the organ break it up into islands of hepatic tissue, each of which may consist of several lobules (*multilobular cirrhosis*), or single lobules (*unilobular cirrhosis*); or the fibrous tissue may run between the cells of the lobules (*intercellular cirrhosis*). The cells are atrophied and mostly stained yellow or brown by particles of pigment. In the fibrous tissue are numerous newly-formed blood-vessels, which can be injected from the hepatic artery, and, in certain cases, numerous double rows of cubical cells, which appear to be small bile-ducts. The organ is at first enlarged by the overgrowth of connective tissue, and very large cirrhotic livers also contain a quantity of fat. The fibrous tissue in course of time contracts, and thus compresses more and more the hepatic cells, the branches of the portal vein, and perhaps the bile-ducts. The liver-cells and fat may disappear, and the organ may be reduced much below its normal weight. The varying size of the liver is thus in part, at least, dependent on the stage of the process.

For the most part the difference above mentioned between livers which are deformed, and nodular or bossy, and those which are smooth or only finely granular correspond to histological differences, and to clinical differences which will be mentioned later. Thus in the former the fibrous tissue separates groups of lobules: it is a *multilobular cirrhosis*. The organ varies much in size: it may be very large, or about the normal size, or very much smaller: in the last case the shape is often much altered from the extensive and irregular contraction of the fibrous tissue. When the surface is smooth, the fibrous tissue separates single lobules from one another, and it is spoken of as *unilobular cirrhosis*. The organ is often very large—indeed, much larger than is common in multilobular cirrhosis; and the shape is little affected. The organ is generally deeply stained with bile, and the fibrous tissue often shows a great number of the double rows of cubical cells above mentioned. Because in the first group of cases the liver is often quite small, and in the second very large, the terms *atrophic cirrhosis* and *hypertrophic cirrhosis* have been employed to distinguish them. But in more than 50 per cent. of cases of so-called atrophic cirrhosis the liver is at the time of death larger than normal, reaching even twice its size; and on the other hand the large livers of so-called hypertrophic cirrhosis may certainly contract to the normal size or below it. I have recorded a case in which a liver which reached below the umbilicus, in a patient with strongly marked jaundice and no ascites, was found fifteen months later to have contracted quite close under the edge of the ribs.

An *intercellular* cirrhosis occurs especially in congenital syphilis, the fibrous tissue invading the lobules and running among the hepatic cells.

Symptoms.—*Multilobular cirrhosis.*—The early stage of cirrhosis often passes with very little disturbance. There may be symptoms of congestion of the organ, such as fulness or pain in the hepatic region, with a slight tinge of jaundice; on the other hand, there is frequently, as a result of free drinking, a gastritis which produces loss of appetite, furred tongue, and vomiting, especially in the morning. An examination of the abdomen at this stage may, however, reveal a considerable enlargement of the liver, of which the patient is entirely ignorant. The next symptom is not infrequently *hæmatemesis*, or vomiting of blood; this is due to the commencing obstruction in the portal circulation: as the blood in the portal vein finds a difficulty in passing through the liver, the radicles of this system, viz., the mesenteric, gastric, and splenic veins, are, of course, congested, and must have a tendency to bleed on the mucous surface of the stomach and intestine. But sometimes the blood proceeds from a rupture of the veins at the lower end of the œsophagus, which have become varicose in the course of establishing a free communication between the portal vein and the inferior vena cava or azygos vein. The quantity thrown up is often considerable, as much as one or two pints; and occasionally the hæmorrhage is directly fatal. The vomiting may be followed or accompanied by melæna. Piles are not infrequently present at the same time.

The most important and constant result of the portal obstruction is the effusion of fluid from the distended veins into the peritoneal cavity, constituting the form of dropsy already described as *ascites* (*see* p. 670). In a large number of cases, when ascites has developed, the liver is still enlarged, and can be felt one or more inches below the ribs; if the fluid be displaced by the hand, as described under Ascites, the surface is felt to be rough, granular, or nodular, and the edge is rounded. If the organ has already contracted below the normal, it may be impossible to feel it; but the results of percussing the lower ribs to estimate the vertical extent of hepatic dulness are always uncertain in such a case, since the presence of fluid allows considerable change in the relations of the liver, intestine, and abdominal walls to one another. The spleen is often enlarged and may be felt. The surface of the abdomen is covered by large veins, running between the iliac and thoracic trunks. This collateral circulation is partly, perhaps, due to the compression of the vena cava inferior by the fluid in the abdomen, for the feet and legs are often œdematous at the same time; but it is also a means by which the portal circulation is relieved. This is an important point, for it must be remembered that the portal system is not completely shut off from the general circulation, but that there are, even in health, means of communication which in cirrhosis become greatly enlarged, and allow of some of the blood in the portal vein radicles reaching the right side of the heart without passing through the liver itself. Those which have been described are communications (1) between the gastric and œsophageal veins at the opening in the diaphragm; (2) between the inferior mesenteric and the hæmorrhoidal branches of the internal iliac vein; (3) between the coronary veins of the stomach and branches of the phrenic veins; (4) between branches of the mesenteric vein and the spermatic vein, or others in the abdominal wall. Frerichs describes (5) vessels forming in the adhesions between the liver and the diaphragm; and (6) a large vein (accessory portal of Sappey) has sometimes been found running along the round ligament of the liver, by which the portal vein communicates directly with branches of the epigastric and internal mammary. Sometimes a loud venous murmur is heard at the epigastrium.

The bases of the lungs are often seriously compressed by a large ascites, and the heart is displaced upwards. The urine is generally scanty, high-coloured, with abundant deposits of red urates, and not infrequently a trace of albumen. This may be due to pressure on the renal veins; it is unsafe to diagnose co-existing granular kidneys from that fact alone.

By the time that ascites is well developed, the patient is in other respects often seriously ill. He is thin, weak, with sunken eyes, a slight tinge of jaundice, and small stellate venules on the face. The temperature is mostly normal, but fever is sometimes present, as shown by Carrington.

The symptoms remain much the same, but the prognosis is very unfavourable. Sometimes recovery follows the use of diuretics and purgatives, and the removal of the fluid by tapping, and is no doubt largely due to the development of the venous anastomoses above noted. But death often results within a few months of the appearance of ascites, from exhaustion, from hæmorrhage, from repeated tappings, from peritonitis after tapping, or, lastly, from cerebral symptoms (delirium and coma), which may ensue at the very time that the fluid is becoming absorbed, and carry off the patient in a few days. This complication must be referred to a toxæmia resulting from the imperfect performance of the hepatic functions.

Unilobular Cirrhosis.—In the cases for which this name has been reserved the earlier indications that alcoholic excess is injuring the liver are not confirmed by ascites, but the main symptom is a more or less intense jaundice. If the liver be examined, it is found to be very large indeed, reaching one or two inches below the umbilicus, giving a certain degree of prominence to the abdomen, and an area of dulness which may measure vertically nine or ten inches. The surface may be smooth or distinctly granular to the touch. The urine is stained with bile-pigment, but the fæces generally retain their natural colour; and the jaundice is attributed to the obstruction of some of the small ducts within the liver. After a time the patient is taken with all the symptoms of acute blood-poisoning; he becomes delirious, even violently so, and relapses into coma. The temperature is high, hæmorrhages occur under the skin and from the mucous membranes, and he dies in three or four days. Whether such a termination is necessary is doubtful. I am inclined to think, from the case above mentioned (p. 682), that if stimulants be withdrawn sufficiently early such livers may contract, when they may be expected to cause ascites in the usual way at some future date.

Diagnosis.—Cirrhosis is often latent until hæmatemesis, ascites, or marked jaundice discloses the secret; it has been already stated that examination may discover an enlarged rough liver in a tippler who has had no decided trouble. Most commonly the diagnosis has to be made when ascites has already appeared, and then the history of drinking and of hæmatemesis, the presence of an enlarged liver, enlarged spleen, and slight jaundice, are sufficient to determine the case. Various conditions of the liver and peritoneum causing ascites have been already mentioned (p. 670). Of these the most important is *cancer*, which may obstruct the portal vein, or its largest branches. The relation of *perihepatitis* to ascites is somewhat uncertain: when the two co-exist there is often also chronic peritonitis and granular kidney. *Cancer* and *tubercle* also cause a peritonitis, which results in ascites. The former may be recognised by the occurrence of nodules of growth in different parts of the abdomen. The latter often presents a thickening of the omentum, which may be mistaken for an enlarged liver. Sometimes the diagnosis can only be made with certainty after a tapping, when the abdominal organs can be better examined. Hæmatemesis is more frequently the result of cirrhosis than of any other disease except gastric ulcer, and is valuable in diagnosis.

When the chief symptom is jaundice, and there is no ascites, the liver is mostly enlarged, and nearly smooth on the surface. The presence of bile in the stools shows that the larger ducts are not obstructed; and tenderness and some febrile action do not exclude cirrhosis. In some cases of cirrhosis and jaundice in children, a very great enlargement of the spleen has been observed, together with stunted growth of the patient and marked clubbing of the finger-ends.

The not infrequent coincidence of multiple neuritis and cirrhosis from alcoholism is worth bearing in mind.

Treatment.—The first essential is that the ingestion of alcohol should be absolutely stopped; and in early stages, where the liver is still uncontracted, and ascites has not yet appeared, it seems likely that the fatal termination may be considerably delayed. The diet should be light and easily digestible; the bowels should be kept active, and sickness and any dyspeptic symptoms may be treated by effervescing salines, bismuth and bitter tonics. But when ascites occurs it is an indication of a serious, and perhaps insuperable, obstruction to the portal circulation: an attempt to promote its absorption must be made by the use of diuretics and purgatives. Of the former, acetate, nitrate, and bitartrate of potassium, spirits of nitrous ether, squills, and digitalis are most frequently given; and the resin of copaiba in doses of 15 grains three times a day sometimes has a beneficial effect. But the kidneys act at a disadvantage from the pressure of the ascitic fluid. Of purgatives, sulphate of magnesium, bitartrate of potassium, compound jalap powder, or elaterium may be employed. If these fail to remove the fluid, and the abdomen becomes very tense, paracentesis is required, and it may sometimes be repeated with success as the fluid re-accumulates. By many, indeed, early and repeated tapping is recommended, as a more satisfactory method than its use at the last moment.

OTHER FORMS OF ATROPHY AND CONTRACTION OF THE LIVER.

Besides the acute yellow atrophy of the liver, and the atrophic form of cirrhosis, some other conditions are described in which the liver becomes very much smaller than in health.

Simple Atrophy.—This occurs chiefly as the result of old age or of inanition. The organ may be reduced to half its normal bulk, but there is no alteration in its structure and no induration, the lobules being diminished in proportion to the size of the whole organ. It produces no symptoms. Partial atrophy of the liver may result from the external pressure of organs in the neighbourhood, or of tight-lacing. Murchison described an atrophy of the liver with a nodulated surface, but without any cirrhosis. Nevertheless, such a liver may cause ascites, and may be indistinguishable from cirrhotic atrophy during life.

Red Atrophy of Rokitansky, or **Chronic Atrophy** of Frerichs.—In these cases the organ is small, without being indurated; it is of dark reddish-brown colour, and contains a good deal of blood. The hepatic cells are atrophied, and contain granules of brown pigment; and lastly, the portal vessels are said to be much dilated right up to their interlobular branches, while the portal capillaries are obstructed by brown pigment, so that they can with difficulty be injected. The causes are said to be malarious fevers and dysentery, or other forms of ulceration of the alimentary canal. It may produce ascites, like cirrhosis; but there is rarely jaundice, and it is to be distinguished mainly by the evidence of atrophy with a smooth surface, and by the history.

Atrophy from Perihepatitis is also said to occur (*see* Perihepatitis).

FATTY LIVER.

The hepatic cells normally contain a small quantity of fat in the form of minute globules. Under certain conditions of disease the fat is immensely increased, and each cell may contain such a large amount that the nucleus and outline of the cell are entirely obscured, and the cell itself might be supposed to be destroyed. This change takes place first at the periphery of the lobule; later the whole is invaded. The liver is much enlarged, it has a smooth surface, is somewhat rounded at the edge, on section has a whitish-yellow colour and uniform appearance, and it may actually float in water. This *fatty infiltration* occurs under two sets of conditions. One is in association with general obesity and fatty degeneration of other organs, such as the heart and muscles. A similar change takes place in some drinkers. The other follows emaciating disease, especially phthisis, and sometimes cancer. In phthisis it may be explained by supposing that the fat cannot be disposed of from defective respiratory powers.

The fatty liver is painless; it can readily be felt as a large, smooth organ in phthisis; but it may be less easy in obesity, on account of the thickness of the abdominal walls. Dyspeptic symptoms and deficient secretion of bile are referred to fatty liver.

A partial form of fatty change occurs as a result of long-continued congestion in the nutmeg liver of heart disease; the fatty infiltration is confined to the periphery of the lobules.

As contrasted with the above deposit of fat in hepatic cells, which still persist intact, an actual *fatty degeneration* with complete destruction of the cell takes place as a result of acute yellow atrophy, and notably in poisoning by phosphorus.

LARDACEOUS DISEASE OF THE LIVER.

(*Amyloid Liver. Waxy Liver.*)

Lardaceous degeneration has been already referred to in connection with empyema and phthisis; and as the liver is one of the organs which is most frequently implicated, a short account of the degeneration must here be given. It consists in the formation in the tissues of a firm, colourless, translucent material, which gives a special reaction with certain colouring agents. Thus, iodine in tincture or aqueous solution turns it a rich brown-red or claret colour. The iodine may be applied to a section of the fresh organ, with the simple provision that it shall be washed free of blood, and the affected parts are then mapped out by the characteristic tint. The subsequent addition of dilute sulphuric acid changes this to a dark purple hue. Methylaniline-violet or gentian-violet turns lardaceous matter red, while the surrounding healthy tissue is stained blue.

The material, which was thought from the iodine reactions to be of a starchy nature, has been shown to contain nitrogen, and to have a chemical composition like the different forms of albumen; the term amyloid is therefore scarcely correct, and the names lardaceous and waxy, which refer to the firm consistence of the organs affected, are at present more suitable.

It resists chemical action and putrefaction. The tissues invaded by it are, first in point of time, the walls of the blood-vessels, especially the inner and middle coats, and the muscular fibres of the same; secondly, various connective tissues; and lastly, if at all, the gland-cells of an organ. That it is not simply a degeneration seems to be shown by the fact that the affected organs are enlarged—that is, that there is more material added than corresponds to what is displaced. It has been suggested that it is due to a transudation from the blood reacting on the substance of the tissue (Ziegler, Payne).

It occurs most often in the spleen, kidneys, liver, intestines, and stomach; and with decreasing frequency in the supra-renal capsules, lymphatic glands, thyroid, aorta, ovaries, and uterus.

Lardaceous degeneration can, in the vast majority of cases, be attributed either to prolonged suppuration from phthisis, syphilis, tubercular disease of bones and joints, and empyema, or to syphilis, without suppuration. Other cachectic conditions have been present in a few instances. The way in which these causes operate is still uncertain. Dickinson's view is that the pus drains off a quantity of alkali from the body, and that the lardaceous material is deposited in consequence. But this is not free from objection when suppuration is the cause, and it has failed hitherto to explain the cases due to syphilis alone.

In the liver the lardaceous change attacks the capillaries of the lobules, and first of all those in the mid-zone, which are most intimately connected with the divisions of the hepatic artery. As this deposit increases the hepatic cells are compressed and atrophied, but they are only occasionally the seat of lardaceous deposit. The liver becomes enormously enlarged, has a smooth surface, a somewhat rounded edge, and is entirely free from pain or tenderness. The disease causes no jaundice. It arises from the several causes mentioned—namely, phthisis, prolonged suppuration, syphilis, or other cachexia; and is accompanied by a similar change in other organs, producing enlarged spleen, albuminuria, and diarrhœa. A lardaceous liver, which is at the same time the seat of syphilitic gummata or cicatrices, naturally loses its uniform smooth surface, but may be recognised by its other associations. The portal circulation is not obstructed by the lardaceous change, and although ascites is not infrequently present, it is mostly associated with general anasarca, and must be referred with it to co-existing disease of the kidneys.

Prognosis.—This is very bad, but decrease of the enlargement after efficient surgical treatment has been recorded.

Treatment.—The cause must be, if possible, removed. This is impracticable in phthisis; but other sources of suppuration may perhaps be treated surgically; and potassium iodide, cod-liver oil, iron, quinine, and other tonics should be given. Mercury and potassium iodide should be used in syphilitic cases.

OTHER ENLARGEMENTS OF THE LIVER.

Enlargement of the liver also occurs in leuchæmia, and a simple hypertrophy is described by authors; but little is known of the conditions under which this last occurs.

SYPHILITIC DISEASE OF THE LIVER.

This may be *congenital* or *acquired.*

Congenital syphilis occurs in two forms: first, as an interstitial hepatitis; secondly, as gumma. The diffuse change begins as a cellular infiltration, which develops into a fibroid induration, and is generally most marked in the interlobular spaces; but sometimes it invades the lobules, and surrounds each cell with a layer of fibrous tissue, constituting a true intercellular cirrhosis, and leading to considerable enlargement of the organ. Probably contraction may take place subsequently. Gummata and cicatrices are less common than in the acquired disease.

Acquired syphilis also produces diffuse hepatitis and gummata, but the latter are much more frequent. They present the general features of gumma in other situations, being more or less circular, yellow masses, tough, and elastic, surrounded by a zone of grey fibrous tissue, from which branch off numerous bands into the adjacent hepatic substance. The contraction of the fibrous tissue tends to produce a depression or fissure on the surface of the liver, at the bottom of which lies the gumma which has caused it; and so the organ may become coarsely lobulated and deformed. Gummata not infrequently break down in the centre into a puriform detritus; on the other hand, they may become completely fibrous, so that nothing remains but a depressed scar; or calcareous granules may be deposited in them. Livers affected with gummata often undergo the lardaceous degeneration, in consequence of which they may be of large size in spite of cicatricial contractions. Perihepatitis is another change resulting from syphilis.

Symptoms.—Occasionally a large gumma may form a prominence on the anterior surface of the liver, smooth and elastic, and strongly suggestive of a hydatid or other cyst. More often, but probably in later stages, syphilitic livers are large, hard, irregular on the surface, and deformed, from the contraction of the fibrous cicatrices. Neither ascites nor jaundice is necessarily present, but in particular cases they may occur from the pressure of a gumma upon the portal vein or the bile-duct. Albuminuria from coexisting lardaceous disease of the kidney is often associated with it. A gumma is sometimes accompanied by decided fever of hectic type.

Treatment.—We may hope for some benefit from potassium iodide in early cases; but in those with old cicatrices and extensive lardaceous disease, little good can result, beyond, perhaps, the prevention of further mischief.

TUBERCLE OF THE LIVER.

This is almost invariably a part of a general tuberculosis. It occurs either as minute greyish-yellow granulations, less than a pin's head in size; or of somewhat larger (3–5 mm.), bright, yellow masses, more easily detached from the surrounding liver-tissue. These larger masses are often softened into a cavity in the centre, which is deeply stained with bile. As a rule, no local symptoms accompany hepatic tuberculosis; but occasionally a general enlargement of the liver results; and in rare cases there is jaundice (*see* Miliary Tuberculosis, p. 137).

NEW GROWTHS IN THE LIVER.

The only tumour of the liver that is at all common is carcinoma. Of others, cavernous angeiomata, simple cysts, and the lymphomatous deposits associated with Hodgkin's disease are the most frequent. They rarely cause definite symptoms. Cases of spindle-cell sarcoma, melano-sarcoma, cysto-sarcoma, myxoma, and adenoma have been recorded.

Carcinoma of the Liver.

Pathology.—Primary carcinoma occurs, according to Ziegler, in three forms—as nodules appearing in any part of the liver, as a diffuse infiltration, and as a growth confined to the interlobular connective tissue. In its structure it often consists of a typical clump of epithelial cells; or they may be formed on a glandular type with cylindrical cells. But more than three-fourths of the cases of cancer of the liver are secondary to cancerous deposits, either in the liver itself or in other organs, especially the stomach and pylorus, the small or large intestine, the gall-bladder, the glands in the portal fissure, the uterus, the female breast, or the vertebræ. A not infrequent antecedent of cancer is the presence of gall-stones in the gall-bladder or in the cystic or common duct. The cancer-cells are carried to the liver by branches of the portal vein, and lodged in the lobular capillaries. The form of the secondary cancer, whether soft or hard, or melanotic, is determined to a certain extent by the nature of the primary growth.

If the cancer is diffused, the liver is merely enlarged; but when it exists in the form of nodules, or separate tumours, the liver takes at the same time the most varied shapes. Each nodule tends to grow evenly in every direction and thus to keep a globular form, and when it reaches the surface, it will project as a hard, convex, or hemispherical outgrowth. But as the nodules become larger—for instance, one and a half to two inches in diameter—they often break down in the centre into granular and fatty detritus, and as a consequence those that project on the surface, being unsupported on one side, sink in and form a central depression or *umbilication*, which is a very characteristic feature of the cancerous liver as felt through the anterior abdominal wall. The lower edge of the liver is also irregular and nodulated. On section, such a liver presents irregular areas of white cancer growth, with a more or less circular outline; the larger ones are softening in the centre, and many of them are blotched by hæmorrhages. The intervening hepatic tissue is often of a deep brown or yellow colour. Where the cancer has started from the gall-bladder, or the bile-duct, or has grown in from the portal fissure, the growth is most extensive in that region, or may be quite confined to it. Sometimes the empty gall-bladder, or a gall-bladder containing some calculi, is embedded in a mass of cancer. Cancer nodules near the portal fissure may compress the bile-duct or the portal vein, and the latter may be entirely filled by the new growth.

Ætiology.—The chief features in the ætiology of cancer of the liver are its relations to the various primary lesions above mentioned, and to gall-stone troubles of older date. Like cancer elsewhere, it is most common between the ages of forty and fifty.

Symptoms.—Cancer of the liver usually gives rise to a good deal of pain, affecting the right hypochondrium, shoulder, and loins. At first not much more than a sense of weight and uneasiness, it afterwards becomes severe and lacinating, and is accompanied by tenderness. Occasionally, however, pain is absent. The liver, as already stated, is enlarged; it may reach far below the umbilicus, and over towards the left side; the nodules are prominent on the surface, and the irregular outline may even be seen in profile. For the most part the enlargement is in a downward direction, but large masses may grow from the convex surface, and force up the diaphragm so as to compress the base of the lung. The surface of the cancerous mass is, as a rule, of almost stony hardness, distinctly more hard than lardaceous disease, or cirrhosis, and the transition from hard cancer to the soft normal tissue can often be recognised. Jaundice occurs in about half the cases, and can generally be shown to result from pressure on the main bile-duct, especially in those cases where the cancer starts from the portal fissure. Similarly, ascites is often, but not always, present, and rarely is the fluid as abundant as it may be in cirrhosis. It mostly depends on direct pressure on the portal vein or its large branches; occasionally on a co-existing peritonitis. The emaciation, pallor, and prostration common to malignant diseases of the abdominal viscera are also present.

Diagnosis.—A jaundice of some months' standing in an old person with an enlarged liver is, in the great majority of cases, due to cancer; though occasionally the bile-duct may be obstructed permanently by a gall-stone. If nodules of cancer can be felt on the liver, the diagnosis is so much the more certain. In cases without jaundice, the large, irregular, and bossy liver, and the emaciation of the patient are generally distinctive. Lardaceous and cirrhotic livers are less hard and more uniform; syphilitic livers may be irregular and painful, but often occur in younger people, and have their own special history. A long history of gall-stones does not exclude, but rather favours the possibility of cancer.

Prognosis.—This is hopelessly bad; the duration is rarely more than twelve months, and the softer forms of growth may kill within a month or two.

Treatment.—This can be only palliative, and consists in relieving pain and in meeting other symptoms, mostly of the digestive organs, such as vomiting, flatulence, and constipation. The diet should be light but nutritious, and, considering the functions of the liver, saccharine and oily substances should be sparingly given.

HYDATID OF THE LIVER.

Hydatid tumours are cysts which contain a colourless, non-albuminous liquid, and which arise as a stage in the development of an intestinal worm, the *Tænia echinococcus.* They may occur in any part of the body, such as the brain, lung, spleen, peritoneal cavity, intermuscular spaces, or spinal canal, but are most frequent in the liver; and accordingly the description of their growth will be given in this place.

The *Tænia echinococcus* is a minute tapeworm, measuring only one-quarter of an inch and consisting of four segments, of which the first has hooklets and suckers, and the last, longer than the other three put together, forms the mature *proglottis* (*see* p. 656).

This worm inhabits the intestines of the dog, and its cystic form infests the sheep, just as the cystic form of the human *Tænia solium* is found in pigs, and that of the *Tænia mediocanellata* in cattle. If the ova of the dog's tænia by any accident reach the human intestine, an embryo in due time escapes, and finds its way to the liver, where it loses its hooks, and is transformed into a vesicle or cyst, containing a clear liquid. Its walls consist of an outer laminated very elastic layer, and an inner, *parenchymatous*, layer, containing granular matter, cells, muscle fibres, and a vascular system. As it grows it sets up a certain amount of irritation in the tissue around it, and a layer of fibrous tissue is developed in immediate contact with it. The liquid is clear, or just opalescent, of specific gravity 1005 or 1007, and free from albumen; but it is said to contain a small quantity of grape sugar, and succinate of ammonium.

As the cyst grows it may be reproductive in three ways. First, when the cyst has reached the size of a walnut, it develops from its inner parenchymatous layer smaller cysts, which remain attached by a pedicle, and in which are formed from three to six or more *scolices*—that is, small cyst-like bodies with four suckers and a ring of hooklets at one end. The cysts containing them are called *brood capsules*, and are sometimes so numerous as to give a velvety appearance to the inner surface of the hydatid cyst. Secondly, the original cyst produces so-called *daughter-cysts*, either directly from the brood capsules which then become detached, or by independent growth between the two layers of the cyst, and subsequent discharge into its interior. The daughter-cysts have the same structure as the original, or *mother-cyst*, and may produce within themselves *granddaughter-cysts*. Thus, a mother-cyst may contain hundreds or thousands of cysts, of all sizes, from a pea upwards. This is called *endogenous* cyst-formation. Thirdly, daughter-cysts formed between the layers may be discharged externally into the liver (or other organ), constituting *exogenous* cyst-formation; and this is more common in animals.

A hydatid-cyst may be sterile, producing neither brood capsules nor daughter-cysts.

A very rare kind of hydatid is the *multilocular hydatid.* It forms a hard globular mass, consisting of a number of cavities or alveoli, about the size of peas, with transparent, jelly-like contents, which Virchow showed to be the remains of hydatid cysts. Scolices are sometimes found, but the cysts are mostly sterile.

The ordinary hydatid cyst may grow to an enormous size, so as to contain several pints of fluid, and it thus constitutes a tumour which exerts considerable pressure on surrounding parts.

Changes in the Cyst.—A hydatid cyst may last for several years, without any essential change beyond its growth; but its existence may be shortened (1) by spontaneous rupture; (2) by death and conversion into a harmless mass; and (3) by suppuration. The exact cause of death is not always clear; it has been thought to be due to the entrance of bile into the cyst, or to the external capsule becoming too rigid to allow of the nutrition of the daughter-cysts; but Fagge mentions cases which are opposed to either view. In any case, the result is that the hydatid is converted into a mass of opaque membranes, more or less closely packed together, and mixed with a yellow pasty or putty-like material, in which calcareous salts and cholesterin crystals can be recognised. Very rarely the contents of the cyst are converted into a gelatinous substance, containing abundance of albumen and chlorides (Bruce and Sheild). If suppuration takes place, the hydatid is also killed, and the abscess thus formed contains shreds of hydatid membrane, and the silicious hooklets from the heads of the scolices. On the other hand, the spontaneous rupture of a cyst is followed by suppuration.

Symptoms.—Of these the most important is the swelling which the liver, enlarged by the presence of the hydatid, forms in the upper part of the abdomen. If the cyst is deeply seated, or on the upper surface of the liver, the swelling may be entirely due to the displaced or enlarged liver; if the hydatid is near the anterior surface, it forms a distinct globular, or hemispherical prominence, which is tense, elastic, and if of sufficient size, distinctly fluctuating. Such cysts sometimes present what is known as the *hydatid thrill.* If the finger or fingers of the left hand are laid on the tumour, and are struck with the tips of the fingers of the right hand, a vibration is set up which can be felt for some little time by those of the left hand which remain applied. It is, however, by no means always present, and it is of doubtful significance, since it is probably due merely to the tension of the cyst-wall, and may therefore presumably be obtained in other cysts, if tightly filled.

Cysts which do not merely project from the lower surface of the liver, but occupy its substance or upper part, frequently exert a local pressure upon the ribs and diaphragm, so that the right side of the chest is markedly bulged, the lower ribs are lifted up, and the costal angle of that side (the angle between the right margin of the ribs and the middle line) becomes larger than that of the opposite side. The base of the lung is often compressed, and dulness occupies the lower part of the right chest, sometimes to be distinguished from that of pleuritic effusion by the margin of its dulness being high in the axillary line, and falling lower as it reaches the spine; whereas the upper margin of dulness of pleural fluid is at the same level all round.

A comparatively small cyst may happen to press on the portal vein and cause ascites, or on the bile-duct and cause jaundice.

Pain is not generally a prominent symptom in hydatid tumour of the liver. It may be entirely absent or it may be severe, even when the tumour is small. It sometimes depends on the occurrence of peritonitis over the cyst, or on the mere size of the cyst causing much local tension. The health of the patient is generally good, and at first entirely unaffected by the presence of the hydatid.

Suppuration of the cyst is commonly indicated by the onset of pain, or its increase if formerly present; the patient loses health and strength, and has elevation of temperature, and perhaps rigors. He has, indeed, an abscess of the liver, with its accompanying conditions; and this abscess may, similarly, point and discharge its contents in different directions. Rupture through the abdominal parietes has occurred with varying results; perforation into the stomach or alimentary canal may be followed by recovery, daughter-cysts and portions of the mother-cyst escaping by the fæces or by vomiting. Or the cyst opens into the base of the lung, and pus, cysts, and bile-pigment are expectorated, also with a favourable result in some cases. Rupture into the pleura, pericardium, or peritoneum is nearly always fatal, the exception being most likely to occur in the case of the peritoneum. Sometimes the hepatic vein, or the inferior vena cava has been opened, and daughter-cysts have been carried into the right ventricle and have blocked the branches of the pulmonary artery; and the portal vein has also been invaded. If the cyst communicates with the biliary passages, bile mixes with the fluid, and stains the daughter-cysts, causing the death of the parasite. Conversely, the cysts may lodge in the bile-ducts and cause jaundice.

Diagnosis.—Hydatid is distinguished by its being a localised swelling of the liver, of long duration, and not affecting the health of the patient. Where it is accessible to palpation, its round, elastic, and fluctuating properties distinguish it from most other enlargements of the liver. Sometimes a hydronephrosis may closely simulate a hydatid, but generally it is distinguished by a lower position, by the colon lying in front of it, and by the slightness of its movement on inspiration. The test of a hydatid cyst is the nature of the fluid drawn from it by aspiration or trocar and cannula. This has the properties already described; in addition, it may contain some flocculi, consisting of detached scolices, or the minute hooklets which are their most characteristic features. These measure about $\frac{1}{1000}$ inch in length, are slightly curved, and present a prominence on the concave side, which makes them almost triangular.

When suppuration has taken place, it is indicated by the local pain, tenderness, and prominence, combined with the constitutional disturbance; and the previous history will generally help to distinguish the hydatid from the tropical or pyæmic abscess.

In the case of a supposed pleuritic effusion, hydatid may be suspected if there is disproportionate displacement of the liver downwards, or if there are other hepatic symptoms. It is proved by the withdrawal of clear hydatid fluid, or thick fluid stained with bile, or of thick grumous or offensive pus containing hooklets.

Treatment.—This is almost entirely surgical in its nature; medicines have no influence upon the growth of the parasite. The fluid may be withdrawn from the cyst by the *aspirator*, and this of itself appears to be fatal to the parasite; at any rate, a cure has often followed this simple proceeding. Fagge states that the re-accumulation of fluid, which is observed after aspiration, is not due to fresh hydatid fluid, but to inflammatory serum, which again after a time subsides, leaving the cyst inert.

But in most cases it seems desirable to make a free *incision*, and insert a drainage tube. Adhesion of the liver to the parietes may be secured before the cyst is opened. Suppuration necessarily takes place, a drainage tube has to be kept in, and the cyst gradually contracts.

In a few cases success has attended the method of *electrolysis*, by means of needles inserted into the cyst and connected with a galvanic battery, but the explanation of the result is not clear.

If the cyst has already suppurated, it must be opened and treated in the same way as an ordinary hepatic abscess.

Prevention.—Since the hydatid of man is obtained from the Tænia echinococcus of the dog, and that is propagated by means of the sheep and pig, it is desirable—first, to prevent dogs from eating offal from sheep and pigs; and secondly, to destroy the tapeworms (or their ova) developed in the dog. For this last purpose it has been recommended to purge dogs periodically, and to burn or bury their excreta; further, to scald frequently the floors of their kennels.

CATARRHAL JAUNDICE.

(*Catarrh of the Bile-Ducts.*)

This is one of the commonest forms in which jaundice occurs. It is generally believed to be caused by obstruction of the common bile-duct by catarrhal inflammation, which has spread up from the duodenum, and which has caused thickening of the mucous membrane of the duct, with or without some excess of its secretion. As the patient almost invariably recovers, opportunities of verifying the diagnosis are quite exceptional; and hence the pathological cause of an apparently catarrhal jaundice may sometimes be open to doubt.

Ætiology.—Catarrhal jaundice is especially frequent in early life. It is often associated with evidences of gastro-duodenal catarrh, but these are not always present. Murchison called attention to a catarrh of the bile-ducts occurring in gouty persons, and occasionally in constitutional syphilis. It may also be set up by local diseases of the bile-ducts, such as gall-stones, but here the jaundice results from the gall-stone, and any symptoms due to the secondary catarrh would be subsidiary. It is usual to associate with catarrhal jaundice the well-known instances of jaundice from fright, the main features of which are, at any rate, similar; and also the cases of "epidemic jaundice," in which it seems likely that a toxic agent is at work. The jaundice of acute illnesses, pneumonia, typhoid, &c., may possibly be catarrhal.

Symptoms.—The patient may have indigestion, weight, pain, or distension of the stomach after food, with, perhaps, occasional sickness for three or four weeks before the jaundice; and in other cases it may occur after unusual indulgence in particular kinds of food; but in very numerous instances the patient knows absolutely nothing of his illness until he himself sees in the looking-glass, or is told by his friends, that his skin is acquiring a yellow tinge. Occasionally, the jaundice is preceded by severe pains in the limbs. A characteristic jaundice then develops, the skin and conjunctivæ are of a bright yellow colour, the urine is yellowish-brown, or as dark as porter, and gives the play of colours with nitric acid; the fæces are pale, or clay-coloured. The temperature is generally normal, and there may be no constitutional disturbance, the patient being able to do his work as usual; but often he is languid, indisposed for exertion, with a bad appetite, and some nausea. There is mostly no pain in the hepatic region, and not even tenderness; but both may be present in moderate degree. The liver, also, is often not at all enlarged, but sometimes its dulness reaches one or two finger-breadths below the margin of the ribs, and the edge may then be felt, as well as the distended gall-bladder. The bowels are variable, most often constipated, occasionally loose. The pulse may be unaffected, but it is especially in this form of jaundice that abnormally slow pulses have been recorded.

The illness lasts from two to five or six weeks, and the jaundice gradually disappears, the urine becoming normal in colour first, and the skin more slowly recovering.

The **Diagnosis** is generally easy. The painless, or almost painless, onset of jaundice in a young person, previously healthy, and presenting no enlargement of the liver, or scarcely any, as a rule, distinguishes it from the jaundice of *gall-stones*, of *cancer*, and of *cirrhosis*, the other most common causes. *Acute yellow atrophy* may begin with a jaundice, which is in no respects different from catarrhal jaundice, and which lasts from three to five weeks before the onset of the serious symptoms. But the disease is extremely rare, and there are never any signs by which one can anticipate its occurrence in a given case.

Prognosis.—With the above exception it is entirely favourable.

Treatment.—The patient need not be confined to bed, nor even to the house, but should take a light, simple diet, and avoid stimulants; and a saline laxative should be given if the bowels are actually confined. Sometimes the jaundice passes off quickly with little else, but in many cases recovery is hastened by the internal use of alkaline remedies, especially the bicarbonate of sodium in combination with rhubarb, taraxacum, or calumba. Sodium salicylate is also said to be useful. Carlsbad and Vichy waters are often recommended, and the Carlsbad salts are especially useful, if a laxative is required. German writers advise daily injections *per rectum* of one or two quarts of water at a temperature of 60° to 90° F., to be retained as long as possible. A recent French writer, M. Chouffard, says that improvement begins almost at once; pruritus and yellow vision disappear quickly, the fæces resume their natural colour, and bile-pigments disappear from the urine in from two to eight days. It is said to be proved that the injections cause contraction of the gall-bladder, and it is suggested that these contractions might overcome any obstruction from accumulated mucus in the common duct. Compression and faradisation of the gall-bladder in cases where this is palpable have also been employed. But the former is not entirely free from risk.

GALL-STONES.

Biliary calculi, or gall-stones, are formed from the bile in the gall-bladder, or very rarely in the bile-ducts in the liver. They vary in size from a mere sand to ovoid masses of two inches in length by an inch in breadth; more often they measure from a quarter to half an inch in diameter. They are often roughly cubical in shape, presenting facets, which indicate that several have been in contact; or they may be more rounded, or spherical. The largest have the ovoid shape, which would result from their occupying the whole cavity of the gall-bladder. They are olive-green, brown, or yellowish-brown in colour, and, on section, often show a central dark nucleus with concentric markings, and radiating lines. The constituents of gall-stones are bile-pigment, cholesterin, and calcium salts. Those which consist mainly of bile-pigment are small, dark, and friable. Others have a nucleus or centre of bile-pigment, and are surrounded by layers of cholesterin crystals which radiate from the nucleus; these stones are generally larger, harder, and have a paler colour. There may be only a single gall-stone of large or medium size, or there may be several hundreds.

Ætiology.—Gall-stones are commoner in advanced life, and occur in women more often than in men. Sedentary occupations and over-indulgence in food seem to have some influence; and the fatty and starchy constituents of food are thought to be more injurious. They are deposited, as a rule, in the gall-bladder, and very rarely in the ducts inside the liver, so that the former must present some conditions of the bile—*e.g.*, of stagnation or concentration—favourable to the deposition of its constituents in the solid form. Cholesterin is likely to be precipitated by anything which lessens the alkalinity of the bile, such as a deficiency of the sodium salts, or a catarrhal state of the gall-bladder or bile-ducts producing excess of mucus, which may undergo acid fermentation.

The bacillus coli communis is found in the bile-passages in association with gall-stones, and may have a share in determining their formation, as for instance, by setting up the antecedent catarrh. It does not normally exist in the bile-passages, but obtains access when the flow of bile is sufficiently retarded.

Effects of Gall-Stones.—They are sometimes entirely harmless, and are often found after death in the gall-bladders of patients who have never been known to complain of their presence; but, on the other hand, they may give rise to very serious symptoms, and are not infrequently the cause of death.

1. One of the commonest results of gall-stones is *biliary colic*, or an "attack of gall-stones," as it is sometimes called. This occurs when the gall-stone is, either by muscular contraction, or by the pressure of bile behind it, driven down into the cystic or common duct towards the duodenum. Here it sets up spasmodic contraction of the duct, and intense pain is the result. The patient is seized, often suddenly, with agonizing pain in the right hypochondrium and lower part of the chest, in the right shoulder, and back. This is often so severe that he is bent double, or writhes on the floor or bed. Shivering may occur, and even epileptiform convulsions, and the patient is pale, collapsed, with profuse sweating, and a small, feeble, generally quick pulse. After a time the pain becomes dull and aching, until a fresh attack of the acuter kind occurs.

The attack may terminate in a short time by the passage of the gall-stone into the duodenum, when the pain subsides. More often, within a few hours, or a day or two of the beginning of the pain, bile-pigment appears in the urine, and the patient becomes jaundiced. This is an indication that the gall-stone is impacted in the common bile-duct, so that an obstructive jaundice has resulted. All the signs of this condition are present

—deep jaundice, bile-pigment in the urine, and pale fæces. This again may end by the passage of the stone into the duodenum, when the bile again flows freely, the pain subsides, and more gradually the jaundice clears up. When this happens, the fæces should be searched for the gall-stone, which may be found by washing them with water, and passing the washings through a sieve.

But the time which elapses between the impaction and the discharge into the duodenum is very variable; it may be several weeks, during which the patient remains jaundiced, and is subject to more or less pain, the liver being also somewhat enlarged, and the gall-bladder distended. Even then the stone may pass, and the patient is free from further trouble until another stone travels down the duct. In this way several attacks may occur.

2. On the other hand, there may be a permanent impaction of the gall-stone in the common bile-duct. This is not infrequently at the termination of the bile-duct in the duodenum, the very small orifice of which offers in the case of rather large stones a greater resistance than the calibre of the duct itself. This continued obstruction has effects both on the liver and on the gall-bladder. The liver is at first considerably enlarged from dilatation of its ducts, which are distended with bile. Sometimes the ducts are dilated uniformly, at others more irregularly into globular cysts; after a time, too, their contents become mucous in character. They exert a certain amount of pressure on the tissue of the liver, and cause it to atrophy; so that subsequently the liver becomes smaller, and rather flaccid. The effect of the impaction on the gall-bladder is to cause its distension, so that it projects below the edge of the liver, nearly in the mammary line; here it can be recognised as a large tense cyst, globular or ovoid in shape (since its fundus is the part that is felt), freely movable and descending on inspiration with the enlarged liver. The contents, which are at first bile, gradually get mixed with mucus, secreted from its lining membrane, and ultimately it may contain this last secretion alone. The same effect upon the gall-bladder will be produced by impaction of a stone in the cystic duct, but the distension is then much greater; and in both cases the much distended gall-bladder may suppurate (*empyema* of the gall-bladder), or its walls may acutely inflame (*phlegmonous cholecystitis*). Sometimes an impacted gall-stone, not large enough to obstruct the duct completely, will cause attacks like ague, probably from nerve-irritation (Ord).

3. In other cases the gall-stones remain in the gall-bladder, and there set up various changes. They may entirely fill it, and can sometimes be felt through the abdominal wall as a hard mass, which may give a sensation of crackling on being handled. They may set up inflammation of the mucous membrane, or suppuration, and

if of large size they cause ulceration or sloughing of the wall of the gall-bladder. The bladder may then contract adhesions to the colon or the duodenum, when a fistulous communication is made between them, and the stone or stones pass directly into the intestine. Such a stone is rarely vomited; more frequently it passes *per anum*, or causes a fatal intestinal obstruction (p. 643). Not infrequently, also, the gall-bladder adheres to the abdominal parietes, and an abscess is formed in them, which communicates often by a sinuous tract with the cavity of the gall-bladder.

4. Amongst the other results of gall-stones are cancer about the gall-bladder and bile-duct spreading to the liver, fatal peritonitis from rupture, ulceration or acute inflammation of the gall-bladder, inflammation of the bile-ducts (*catarrhal and suppurative cholangitis*), and formation of abscess in the liver (*see* Abscess of the Liver, and Pylephlebitis). The bacillus coli communis appears to be an important agent in some of these suppurative processes. Though the different forms of cholangitis and cholecystitis are mostly the results of gall-stones, they may arise in connection with cancer and hydatid disease, and acute cholecystitis is an occasional complication of enteric fever. Suppurative cholangitis constitutes one form of febrile jaundice; and acute cholecystitis may lead to acute peritonitis, of which the cause may only be recognised by the situation of the earliest pain in the right hypochondrium, or by the history of gall-stones.

Diagnosis.—Biliary colic may be confounded with other sources of pain in that region: pleurodynia, pleurisy, intercostal neuralgia, gastric pain, intestinal and renal colic, which can generally be distinguished by the localisation of the pain or other features accompanying them. But the passage of gall-stones is not always associated with pain, and cases of impaction with jaundice may be misunderstood from the absence of this symptom. In older cases, the history of repeated attacks is of great service. The recognition of the inflammatory complications described must depend on a careful analysis of the symptoms.

Prognosis.—This need not be unfavourable in a first attack of colic; many people recover even after several; but when jaundice from impaction is of long duration, the possibility of more serious consequences, such as cancer, must not be forgotten.

Treatment.—Any special treatment for the prevention of gall-stones can scarcely be undertaken before their presence is indicated by an attack of biliary colic. The diet should then be carefully regulated, moderate in quantity, starch and fat should be limited, sufficient exercise should be taken, and alkaline waters should be drunk freely. Those of Carlsbad, Vichy, Kissingen, Marienbad, and Ems are especially recommended. Sodium sulphate (1 to 2 drachms daily), sodium bicarbonate (20 to 30 grains), sodium phosphate (1 drachm), and olive oil (2 to 10 ounces) have been recommended for the prevention or solution of gall-stones, but their value is doubtful.

For an attack of biliary colic, the patient should be placed in a hot bath, or hot fomentations or poultices should be applied to the right side. Most relief will be obtained from the subcutaneous injection of morphia in a dose of a quarter to a third of a grain, to be repeated, if necessary, in three or four hours; similar relief, but less speedy, will be given by opium, beginning with 2 grains, and giving another grain every three hours till relief is obtained. Murchison recommended half a grain of extract of belladonna every two hours; and chloroform may be inhaled with temporary relief.

The effects of gall-stones on the gall-bladder and surrounding parts may have to be treated as they arise, and according to the symptoms which they produce; but the limits of treatment have been considerably extended of late years by the surgical operation of *cholecystotomy*. The abdominal wall is incised, the gall-bladder is opened, mucous or purulent fluid and gall-stones are removed, and the edges are stitched to the abdominal wall. The fistula heals within a few weeks. The removal of the gall-bladder (*cholecystectomy*) is desirable in some cases.

PERIHEPATITIS.

Pathology.—Perihepatitis, or inflammation of the capsule of the liver, may be acute or chronic, localised or more generally diffused. It is set up by several of the structural changes in the liver, such as cirrhosis, lardaceous degeneration, syphilitic disease, cancer, hydatid, and abscess; by extension of inflammation from an ulcer of the stomach, and from pleurisy, and it forms part of a general peritonitis. But in some of the most marked cases the cause is by no means obvious. It is not due to spirit-drinking. Fagge showed its frequent association with Bright's disease, and considered that if there was a disproportionate amount of ascites in a case of renal dropsy, it was likely to be due to perihepatitis.

When the liver is affected with perihepatitis, the capsule is opaque and more or less thickened; often the thickening is distributed in patches irregularly over the surface; and such patches may be determined by the disease which causes the inflammation of the capsule. Sometimes the liver is completely enclosed in a membrane, one or two millimetres or more in thickness. In such cases the organ has a rounded anterior edge, which is due to the actual margin of the liver being bent back upon the upper surface, and held there by the thick capsule. This membrane may be stripped off without destroying the tissue beneath. The consistence of the liver appears to vary; Fagge says it is rarely cirrhosed, but soft and often fatty. Murchison spoke of induration arising from perihepatitis. In these severer forms ascites is generally present; which has been supposed to arise either from constriction of the portal vein in the portal fissure, or from compression of the whole organ by its thick capsule, so as to hamper the portal circulation, or lastly to be independent of the liver, and to result from the accompanying peritonitis (Hale White). The spleen is often at the same time similarly affected (*perisplenitis*).

Symptoms.—In some cases of perihepatitis, probably more often those in which the process is acute or subacute, and brought on by the changes in the liver above enumerated, there are other definite symptoms of its presence—namely, pain—which Murchison describes as constant and severe, aggravated by pressure, movement, or coughing, and increased by lying over on the left as well as on the right side. With this there may be some fever, but jaundice is not often present. Occasionally, a friction sound can be heard, or the rub can be felt on laying the hand over the hepatic region. To this cause no doubt is due some of the pain of hydatid, cancer, abscess, and other hepatic diseases.

Treatment.—Pain must be relieved by opium, and local anodynes. Paracentesis may be necessary for ascites.

PYLEPHLEBITIS.

This occurs in two forms, adhesive and suppurative, which have been already referred to, the one as a cause of ascites (p. 670), the other in connection with multiple abscesses of the liver.

ADHESIVE PYLEPHLEBITIS.

This is more generally a thrombosis of the portal vein, in which the clot adheres to the wall of the vein, and becomes ultimately organised in the same way as thrombus in other situations. Its causes are those changes which bring about retardation of the blood-current in the portal vein or its distribution, such as cirrhosis, syphilitic disease, the pressure of tumours on the trunk of the vein, or its implication in perihepatitis, or chronic peritonitis near the fissure of the liver.

Symptoms.—The obstruction to the portal vein necessarily leads to symptoms closely resembling, or identical with, those of cirrhosis—viz., engorgement of the portal vein radicles, showing itself in ascites, with enlarged spleen, diarrhœa, and hæmorrhage from gastric and intestinal vessels. The collateral circulation becomes developed in the same way as in cirrhosis, and the superficial abdominal veins are commonly enlarged, relieving thus, for a time, the portal vessels. The urine is scanty. Jaundice is rare, and only occurs as a result of that which causes the pylephlebitis. The liver is generally smaller than normal.

The **Diagnosis** from cirrhosis of the liver is generally difficult. It must depend on the absence of alcoholic history, or the known presence of conditions which might cause portal thrombosis; but most of these conditions are themselves possible causes of direct portal obstruction and ascites.

Its **Treatment** is similar to that of cirrhosis.

SUPPURATIVE PYLEPHLEBITIS.

This is generally secondary to inflammation in some part of the area from which the portal vein arises, such as ulceration of the appendix cæci, ulcer of the rectum, dysenteric ulcers of the intestine, gastric ulcer, suppuration of the mesenteric glands, or abscess of the spleen. Nearer the liver, gall-stones may set up inflammation of the portal vein branches, or the same may happen from suppuration about a hydatid cyst. In the new-born the portal vein may be the subject of septic phlebitis through the umbilical vein.

The mischief commonly begins either in the branches or in the tributaries of the portal vein, rather than in the trunk itself, to which, however, the suppurative process may ultimately extend. The wall of the vein inflames and suppurates, a thrombus forms in the neighbourhood, breaks down into pus, and in its conveyance to other parts of the vessel sets up fresh centres of thrombosis, phlebitis, and suppuration. Finally, in many cases, multiple abscesses of the liver are formed.

The liver is enlarged, soft, flaccid, and anæmic. The branches of the portal vein are filled with disintegrating thrombi, or pus, or grumous fluid; and the walls of the corresponding veins are infiltrated, or ulcerated. The spleen is enlarged, and there is occasionally peritonitis.

The **Symptoms** are nearly the same as those of multiple abscesses. There are epigastric and hypochondriac pain, fever of hectic type, rigors, sweating, vomiting, and prostration. The portal vein may be sufficiently obstructed to cause some ascites, and the spleen is enlarged, partly on this account, partly as a result of septic fever. Jaundice is often, but not always, present: and, if abscesses are numerous, there may be enlargement of the liver. The fæces are generally coloured. A typhoid condition supervenes with stupor and delirium.

Diagnosis.—The disease may possibly be confounded with pyæmia, septicæmia, intermittent fever, acute yellow atrophy, or tropical abscess. Its recognition depends upon the exclusion of sources of general pyæmic infection, the presence of fever with rigors, the local evidences of the liver being involved, such as pain and swelling, and the evidences of portal obstruction, such as diarrhœa, and enlargement of the spleen.

Prognosis and **Treatment.**—The disease runs an acute course, is generally fatal, and can only be treated by relieving symptoms.

DISEASES OF THE PANCREAS.

The pancreas is subject to pathological processes similar to those that occur in other organs; but it is rarely that its lesions are recognisable by well-marked clinical features. For either the symptoms are obscure, or they are referred to adjacent organs, such as the liver, stomach, and duodenum. There is an important connection between glycosuria or diabetes mellitus and disorders of the pancreas, both acute and chronic.

Pancreatitis.—Acute inflammation of the pancreas sometimes occurs in infective diseases, such as enteric fever, pyæmia, and septicæmia; or it may arise by extension from neighbouring parts. In enteric fever it is commonly of the parenchymatous form, the connective tissue being infiltrated with leucocytes, and the gland-cells in a state of cloudy swelling.

An *acute hæmorrhagic pancreatitis* is described (Fitz) in which the organ is filled with blood, and perhaps the surrounding structures also. The symptoms are sudden acute pain, tenderness, muscular rigidity, nausea, vomiting, and collapse, with a close resemblance to intestinal obstruction or perforative peritonitis. After a few hours a circumscribed swelling may appear in the epigastrium.

In *suppurative* forms the organ is large, swollen, and infiltrated with pus; or it contains separate abscesses. The symptoms are similar to those of the hæmorrhagic form, but less definitely pronounced. *Gangrenous* changes may be associated with the hæmorrhagic: they often lead to peritonitis.

Post mortem the pancreas is found to be swollen, especially its head; and blood, pus or gangrenous changes are present. *Fat-necrosis* is also not uncommon in the pancreas, as well as in the sub-peritoneal tissues around.

Chronic Pancreatitis affects the interstitial tissue, producing considerable fibrous growth, with consequent atrophy of the glandular structures, analogous to the changes in cirrhosis of the liver. The head of the organ is usually most affected. It commonly arises from the spread of adjacent inflammations, such as those of the peritoneum, of the bile-duct, or of a gastric ulcer. It also results from the presence of concretions in the pancreatic duct, or of retained pancreatic secretions; from the irritation of cancer; from the venous congestion of heart disease; and possibly from syphilis, and the abuse of alcohol. Chronic inflammation is one of the changes which are sometimes found in association with diabetes. The symptoms are rarely prominent. Occasionally, on account of diminution or loss of the pancreatic secretion, the fatty constituents of the food are imperfectly digested, and *fatty stools* are passed, in which an oily liquid is actually present, or lumps of white or yellow tallow-like fat. This may occur in any severe chronic lesion of the pancreas; but it is not constant, since the bile assists in the solution of fatty matters, and it has already been shown that fatty stools may occur when the biliary secretion is retained. Dr. Walker, of Peterborough, has brought forward cases to prove that the absence of the pancreatic secretion causes the stools to be clay-coloured, although the bile is normally excreted; and this he explains by supposing that the colouring matter of the fæces is only formed as a result of the co-operation of the pancreatic secretion with that of the liver.

Atrophy, Fatty Degeneration, Fatty Infiltration, and **Lardaceous Degeneration** also affect the pancreas. Acute and chronic inflammation, atrophy and fatty disease of the pancreas have been found in some cases of diabetes, but the connection between the two diseases is not yet clear.

Hæmorrhage sometimes takes place as a result of the congestion of heart disease, and blood-cysts may form in the substance of the gland. A few instances are on record where hæmorrhage seemed to be the sole cause of death.

Pancreatic Concretions.—These consist of calcium carbonate and calcium phosphate. They may be like grains of sand, or as large as hazel-nuts, and are usually round or oval, occasionally irregular or branched. In colour they are white, or greyish-white; sometimes brown or nearly black. They sometimes block the duct or its branches, and lead to dilatation of the ducts, retention-cysts, acute inflammation with suppuration or chronic induration, and even to inflammation in the parts around. They rarely produce symptoms, except through their secondary effects—for instance, by the inflammation which they excite, or by the formation of cysts.

Tumours.—Of these *carcinoma* is the most important, whether primary or secondary. Sarcoma, tubercle, and gumma only occasionally occur. Primary cancer is generally of the scirrhus variety, forming hard nodules in the head of the organ, to which part it is often confined. An irregular nodular hard tumour is thus formed, which may be of sufficient size to be felt under favourable circumstances through the abdominal parietes. As the cancer nodules increase in size the pancreatic duct is liable to be obstructed, with the formation of cyst as a result; and the common bile-duct is not infrequently blocked either by pressure or by the spread of a chronic inflammation, so that jaundice is produced. This is, indeed, a common cause of jaundice in persons of middle and advanced age. In other instances the cancer may involve the stomach, duodenum, peritoneum, vertebræ, or other structures. The symptoms in cancer of the pancreas are deep-seated pain, of aching, gnawing, lancinating, or burning character, which is often distinctly paroxysmal in its occurrence, and is affected by food, coughing, deep breathing, movement, or posture. Nausea and vomiting may be present; and sometimes fatty stools. Examination may reveal a tumour, of the characters described, in the situation of the head of the pancreas. In the later stages emaciation, anæmia, and prostration become prominent features of the case.

Treatment is mainly directed to the relief of symptoms. Removal of some tumours by operation may be possible.

A *cyst* of the pancreas may reach a considerable size. It forms a globular tumour in the upper part of the abdomen, behind the stomach and the colon. The surface is dull or resonant, according to the extent to which it is covered by either of these hollow viscera. The fluid within it is turbid, brown or greenish in colour, alkaline, of sp. gr. 1010–1020; it contains albumen, sugar, mucin, and a trace of urea; and it has the property of digesting starch and emulsifying fat. Hæmorrhage may take place into it. There is emaciation, and sometimes pain or jaundice. The swellings most likely to be confounded with it are a hydatid cyst of some other organ, hydronephrosis, circumscribed peritonitis, and ovarian disease. The nature of the aspirated fluid should help. The treatment is surgical—namely, incision and drainage.

DISEASES OF THE PERITONEUM.

PERITONITIS.

The peritoneum lining the surface of the abdomen, and covering nearly all the viscera contained within it, is liable to inflammation from a number of causes originating in these organs. This inflammation may be acute or chronic, and general or circumscribed.

ACUTE PERITONITIS.

Ætiology.—The most frequent cause is some lesion of the abdominal viscera or adjacent parts, such as ulceration of the stomach, typhoid and tubercular ulcers of the ileum, dysenteric ulcers of the colon, inflammation and sloughing of the appendix cæci, abscess of the liver, suppuration of the gall-bladder, infarction and abscess of the spleen, the numerous inflammatory lesions which are apt to involve the female pelvic organs—metritis, ovaritis, suppuration of the Fallopian tubes, pelvic cellulitis, and pelvic hæmatocele.

In many of these cases the peritonitis is set up, not so much by extension of inflammation, as by the discharge into the abdominal cavity of liquids, such as food, fæces, or pus, which owe their irritating properties in large part to septic organisms. This happens in the case of the perforation of gastric and intestinal ulcers, in appendicitis, and in rupture of abscesses. Peritonitis is the natural termination of most cases of intestinal obstruction, either from local inflammation, as in acute strangulation and hernia, or from rupture of over-distended gut, as in the more chronic strictures. Perinephritic and psoas abscesses may rupture into the peritoneum, and empyema occasionally sets up inflammation below the diaphragm, though it is much less common than pleurisy and empyema as a result of a peritoneal abscess. Wounds of the peritoneum, whether from injury or surgical procedure, are liable to be followed by peritonitis.

As to more general causes, it is not quite clear how far they operate. Cold is probably never the sole cause of peritonitis. If peritonitis forms part of a general septicæmia or pyæmia, puerperal or otherwise, it probably arises directly from local lesions. But Bright's diseases, both acute and chronic, are certainly occasional causes of peritonitis, which is then generally fatal.

Morbid Anatomy.—The changes which take place in the peritoneum are not unlike those which occur in the pleura when it is inflamed. There is at first redness from increased vascularity, and if the cavity of the abdomen is examined in this early stage, the redness on the intestine is commonly seen to be most marked along two parallel lines, which are determined by the diminished atmospheric pressure in the space between any two coils and the abdominal wall (hence called suction-lines by Moxon). This injection is soon followed by the effusion of lymph or pus. There is at first a mere stickiness of the peritoneal surface, but the lymph soon becomes more abundant, forming yellowish flakes, coating the surface of the bowel, or collecting in larger masses in the angles and sulci between the coils. With this lymph, which consists of fibrin and leucocytes, there may be a varying amount of turbid serum. The rapidity with which this forms is very remarkable, as may be seen in some traumatic cases, where a quantity of yellow lymph may be found in less than eighteen hours. In some less severe or less extensive cases, the lymph may develop into fibrous tissue, by the growth of some leucocytes into fibres, and of others into blood-vessels, and the different viscera are united together, or the peritoneal cavity is obliterated by the adhesions which are thus formed.

In other cases, the quantity of leucocytes increases, or is more numerous, from the first, and the inflammatory products are entirely purulent; this is often quickly fatal, but occasionally a large peritoneal abscess may slowly form, and offer chances of recovery.

Acute peritonitis is sometimes, from the first, circumscribed, and results in a localised abscess, which may point externally or open into one of the hollow viscera. Such abscesses occur in the pelvis, or between the diaphragm and the liver, or between the diaphragm and the spleen. In these last two situations they may rupture into the chest, and set up pleurisy or pneumonia. A peritoneal abscess not infrequently contains air, either from direct communication with the stomach or intestine by perforation of an ulcer, or from decomposition induced by contact with the coats of the bowel.

Micro-organisms have an important influence in the causation of peritonitis. When the peritoneum is infected from the intestine, as in appendicitis or perforation of the bowel, or from the biliary passages, the bacillus coli communis is found; in peritonitis derived from without, whether from lesions of the pelvic organs, or the abdominal walls, streptococci and staphylococci are present. The tubercle bacillus is, of course, responsible for the tubercular form (see p. 714).

Symptoms.—*Acute general peritonitis* begins with pain, which is mostly very severe, and, if at first localised to one spot, soon becomes diffused over the whole abdomen. The pain is constant, but aggravated by every kind of movement, by coughing, straining, or vomiting. It is not relieved by pressure; on the contrary, there is marked tenderness over the whole of the abdomen. Vomiting, as a rule, soon sets in, and occurs repeatedly, either spontaneously or after attempts to take food. At first the gastric contents are brought up, subsequently bile, and later still, in some prolonged cases, the vomited matters may have an almost feculent character. The temperature commonly rises, reaching 102° or 103°, more rarely 104° or 105°, and the pulse is quick, 100 to 120; sometimes, also, rigors occur at the commencement; but there is always a considerable degree of collapse.

In some cases of perforation of gastric or intestinal ulcer, death occurs from collapse in twenty-four hours. In others, the patient is soon obliged to take to his bed, and within a short time lies on his back, with shrunken face, dark sunken eyes, anxious expression, dry furred tongue, and quick small pulse. The legs are frequently drawn up to prevent stretching of the abdominal parietes, and every movement is avoided by the patient. The abdomen is at first tense, with rigid muscles, and generally becomes swollen from paralysis of the muscular coat of the intestines, and the accumulation of gas within them. The surface is resonant, but if much fluid is poured out, it may cause dulness at the flanks, or occasionally all over. Gas may escape into the peritoneal cavity, and cause extensive resonance, or even splashing from mixture with the liquid. The extravasation of gas into the peritoneum may sometimes be recognised by its lying in front of the liver, and replacing the natural hepatic dulness by resonance. But it must be remembered that the liver may be displaced from its contact with the anterior thoracic wall by much gaseous distension of the viscera, without any escape of gas from their interior. The tension of the abdomen causes pressure on the under surface of the diaphragm, with consequent dyspnœa, and generally the respiratory movements are entirely thoracic. Hiccough is also a frequent symptom. The bowels are, as a rule, confined; sometimes, after two or three days, one or more motions may be passed, or even diarrhœa may set in; and occasionally there is diarrhœa throughout. The urine is scanty; it may be passed with pain, or be retained.

The patient gradually gets exhausted by vomiting and pain; the tongue becomes drier and brown; sordes form on the lips and teeth; the pulse is smaller and quicker; the bases of the lungs are compressed; and after an illness of from two to six days, death takes place. It is not, however, every case that presents all the characteristic signs. Fever may be absent in some cases; there may be but little distension in others; occasionally, a patient, instead of lying prostrate on his back, will throw himself about in the agony of pain.

In *acute circumscribed peritonitis*, the symptoms are much the same, but the local conditions are more or less limited to the region affected. If pus forms, a more or less defined tumour may be recognised, and fluctuation may be detected. The constitutional symptoms are often less severe than in general peritonitis; and the case may possibly run on into a chronic stage. The fever is variable. The later course of the illness is determined by the behaviour of the inflammatory products: a sero-fibrinous peritonitis may subside; a peritoneal abscess may burst into different cavities, or set up inflammation in the chest, in which case the symptoms of pneumonia, pleurisy, empyema, or pneumothorax may complicate the abdominal lesion, rendering a fatal result highly probable, but not absolutely certain. Disease of the appendix cæci, and lesions of the uterus and its appendages, are the more common causes of acute circumscribed peritonitis.

Subphrenic pyo-pneumothorax.—This is the condition which results from a local peritonitis in the upper part of the abdomen, accompanied by the escape of air into the peritoneal cavity. The most common cause is the perforation of an ulcer of the stomach; hence the abscess is mostly on the left side. The air-containing cavity then lies between the left lobe of the liver, and the left half of the diaphragm, and is bounded on the right by the falciform ligament, and in other directions by the anterior abdominal wall, the stomach, and spleen. On the right it is between the upper surface of the liver and the diaphragm, and is limited to the left by the falciform ligament. The cavity contains pus and air; the liver and spleen are depressed, and the diaphragm is pushed upwards so that a pleural pneumothorax is simulated by the occurrence of tympanitic note, amphoric breathing, metallic tinkling, and bell-sound.

Diagnosis.—As a rule, this is not difficult: the severe pain, tenderness, vomiting, distension, constipation, and collapse form the important features. But peritonitis may be simulated by the severe pain of colic, by hysteria, or by ruptured aneurysm; it may itself be mistaken for intestinal obstruction, and it may be set up and cause death without its presence being suspected, in enteric fever, and after operations on the abdominal walls, such as herniotomy. *Colic* and *hysterical pain* are mostly to be distinguished from peritonitis by the contracted abdomen and the absence of tenderness—indeed the relief on pressure, in the former case; and the extreme sensitiveness to the merest touch, without pressure, in the latter. Indications of lead-poisoning in the former, or a history of hysterical attacks in the latter, would assist the diagnosis. A high temperature of extreme collapse would be in favour of peritonitis. A *ruptured aneurysm* would cause pain and collapse, and might be mistaken for the perforation of a gastric ulcer, and the antecedents in the two cases might be difficult to discriminate. Another condition that has given rise to a diagnosis of perforation and peritonitis is the fatal coma or collapse of *diabetes*. This often begins suddenly, with severe abdominal pain accompanied by collapse, and small, thready pulse. Most such cases are known to have diabetes, or a history of its symptoms can be elicited. Peritonitis resembles *intestinal obstruction* in its pain, distension, vomiting, and constipation, which may hamper the diagnosis for two or more days; the generally diffused tenderness, early distension, and simply gastric or bilious vomiting, are in favour of peritonitis; and this is often at length confirmed by the passage of fæces. The onset of peritonitis in typhoid fever may be very insidious; the patient is perhaps semi-comatose or delirious, and his senses are dulled; the abdomen is already much distended and tense. On the other hand, the tympanites and pain of this fever may be considered due to peritonitis when none exists.

As to the differential diagnosis of peritonitis, its cause must be looked for in the preceding history. Where severe fatal peritonitis ensues in a person previously considered well, ulceration of the appendix cæci, gastric ulcer, and lesions of the pelvic organs are the most likely causes. The first is more probable in both sexes before and about the ages of puberty; the last occurs almost exclusively in females. Perforations of the intestines, whether typhoid, tubercular, or dysenteric, are generally preceded by recognisable illnesses; but, as is well known, typhoid may be of such a mild nature as not to attract sufficient attention on the part of either friends or medical man.

Subphrenic pneumothorax has to be distinguished from *pleural pneumothorax.* In the former there is often the history pointing to gastric disease, there is no cough or expectoration, the heart is only slightly displaced and that upwards, there is no intercostal bulging, the liver and spleen are less depressed, and the diaphragm in some cases moves freely. The diagnosis from *gaseous distension of the stomach* by physical signs is difficult, but the associated conditions would be different.

Prognosis.—General peritonitis is a very fatal disease. The result must be estimated by the character of the pulse, the persistence of vomiting, the amount of collapse, and the probable extent of the inflammation. Severer cases can only be judged of from day to day. There is more hope when some days have elapsed, but in cases that are apparently improving, accumulations of pus may reveal themselves, and become dangerous in the way indicated.

Treatment.—Certain cases of peritonitis, as for instance, those due to perforation of the stomach, or sloughing of the appendix cæci, or other similar accident, are only likely to recover if promptly treated by surgical methods—the abdomen being opened, the cavity being washed out, and the causative lesion dealt with. The same may be said of general suppurative peritonitis, or localised peritoneal abscesses. One of the first considerations then is, whether the case is one of this kind.

But there are cases of adhesive general or local peritonitis, in which surgery is not required. The first principle of treatment is to keep the intestines completely at rest. For this purpose the patient must, of course, be in bed; food should be given by the rectum; purgatives should be strictly avoided, and opium should be administered. The patient's thirst may be quenched by small pieces of ice sucked from time to time, but no food should be allowed by the mouth; the enemata may be peptonised beef-tea, milk and egg to the extent of four ounces given every four hours, or nutrient suppositories may be employed. Opium should be given in full doses: 1 grain in pill every three hours is a quantity frequently used, but it may be given more frequently if required by the severity of the pain, as for instance, one grain every two hours. In such a case the closest watching on the part of the physician is necessary, and the respiration should not be allowed to go below thirteen or twelve in the minute. Morphia may be used instead of opium, one-third or half a grain may be injected subcutaneously at first, and doses of a quarter of a grain may be given from time to time so as to control the pain.

Relief is afforded locally by the application of hot linseed-meal poultices, or flannels wrung out of hot water, and sprinkled with turpentine, or liniment of belladonna. Ice compresses or pieces of ice between layers of flannel are sometimes used, but they do not generally give so much relief as the hot applications. If there is much distension with gas it may be possible to draw some off by passing a long tube into the rectum. Stimulants are, as a rule, required, and are best given in the form of brandy, in small quantities frequently.

CHRONIC PERITONITIS.

This may arise as a sequel of acute peritonitis, especially in its local forms; it is often the result of local irritation about particular organs—for instance, the liver or the spleen may be surrounded with a thickened capsule (perihepatitis, perisplenitis); or it may occur after repeated tappings for ascites. The growth of tubercle and cancer in the abdominal cavity gives rise to forms of chronic peritonitis, which will be spoken of shortly. Amongst general causes, Bright's disease is the best recognised; often it is impossible to explain its occurrence.

Morbid Anatomy.—Chronic peritonitis, in its lesser degrees, causes no more than thickening, with opacity of the peritoneum; in more advanced cases, it results in the formation of false membranes, or layers of fibrous tissue, which cover the different viscera, and cause them to become adherent to one another, and to the abdominal wall. The intestines may thus be matted firmly together, and bound down towards the spine; the omentum is shortened and contracted into a transverse band at the upper part of the abdomen; and the liver and spleen may be covered with a thick adventitious membrane. This membrane can sometimes be peeled off, leaving the natural serous layer beneath. Serous fluid may be present at the same time in sufficient quantity to cause enlargement of the abdomen; or there may be chronic effusion, with but little false membrane, only some opacity or pigmentation of the peritoneum. As already mentioned, chronic peritonitis may result in intestinal obstruction.

Symptoms.—These are very variable, according to the extent of the abdominal lesion. There is usually some pain, or a sensation of tightness and oppression. The appetite is likely to be indifferent, and occasionally vomiting may occur. The bowels are generally constipated. Constitutional disturbance may be but slight, but fever, if present, is variable, and often the patient is about, though unequal to much exertion. The condition of the abdomen depends on the nature of the effused products; if serum exists in any quantity, the abdomen is enlarged, and gives the sense of fluctuation on percussion. Sometimes there is dulness in the flanks, and resonance in front, with change of relative position of these signs when the patient lies on one side; in other cases, the abdomen is completely dull from the intestines being bound down to the spine. When there is little or no fluid the abdomen may be flat, and it presents irregular resistance where the intestines are matted together.

Diagnosis.—Cases with much effusion resemble ascites, such as that produced by hepatic cirrhosis; in women, if the surface is completely dull from the intestines being bound down, the peritoneal fluid may be mistaken for that of an ovarian cyst; in other cases, the resistance felt in the abdomen may be confounded with different forms of tumour.

Prognosis.—This is, on the whole, unfavourable in cases with pronounced symptoms.

Treatment.—There is not the same necessity for absolute quiet on the part of the intestines as in the case of acute peritonitis; food should be light and nutritious; pain may be relieved by poultices and hot fomentations, and by opium in small doses. The absorption of the effused products may be attempted by the application to the abdomen of mercury in the form of compound ointment, liniment, or oleate; while iodide of potassium and iodide of iron may be given internally, with general tonics, such as quinine, cod-liver oil, or arsenic. Constipation may be relieved by enema, or by the mildest laxatives. Large accumulations of liquid require paracentesis.

TUBERCULAR PERITONITIS.

The actual inflammation in this case is preceded by the formation of tubercle as a result of the invasion of the bacillus. The surface of the peritoneum is covered with small, flat, whitish grains, from 2 to 5 mm. in diameter, slightly elevated above the surface, and closely aggregated together. These tubercles are most abundant on the under surface of the diaphragm and in the flanks. The inflammation is shown by the effusion of lymph, or of serous or sero-fibrinous liquid. In very acute cases lymph alone may be present. In less acute cases there is liquid, which may amount to several pints, and the abdomen may be as much enlarged as it is in ascites from cirrhosis or heart disease. More rarely the liquid is sero-purulent or purulent. In other more chronic cases the results are varying degrees of caseation, fibrosis, and ulcerative destruction. Thus, the omentum tends to become contracted into a thick mass, consisting of tubercular and caseous infiltration. The intestines are matted together and adherent to the omentum or the abdominal wall, and the adhesions may be much infiltrated, so as to present thick masses of almost tumour-like new tissue. In cases where ulceration of the bowel co-exists, the adherent intestines may open into one another through the bases of the ulcers, and render it impossible to follow the natural course of the bowel. More or less turbid serum or pus may be present in these cases also; and the mesenteric glands are often caseous also (*see* p. 738).

Ætiology.—Tubercular peritonitis occurs at all ages, but appears to be more frequent in males than in females. It is very commonly associated with tubercle in other parts of the body. It is hence often secondary to pulmonary phthisis, to tubercular ulceration of the intestine, to caseous mesenteric glands, and to diseases of the pelvic organs—*e.g.*, the Fallopian tubes, or the testes and vesiculæ seminales. It may form part of an acute general tuberculosis. Sometimes, perhaps, the peritoneum is infected directly from the bowel. Not infrequently other serous membranes, the pleura and the pericardium, are simultaneously affected.

Symptoms.—The symptoms are sometimes *acute* and the case is similar in every respect to acute peritonitis from other infections. More often they resemble those described under *chronic* peritonitis. They have come on slowly and insidiously, consisting of pain and discomfort, and frequently distension of the abdomen. The patient loses strength and flesh; there is irregular pyrexia; the appetite is diminished, and the bowels are irregular, but often loose. The motions are sometimes yellow, and in acuter cases may suggest enteric fever. The abdomen is generally tender. If there is much fluid effused the abdomen will be much enlarged, tense, resonant in its upper part, or dull all over according as the intestines are free or bound down. More often there is but little fluid in the abdomen, which is tender, slightly swollen, very tense, presenting to palpation increased resistance in certain parts, or a doughy sensation; or there are firm rounded masses with a more or less definite outline. Such tumour-like masses often occupy the lower half of the abdomen, reaching perhaps higher on one side than on the other: they are irregular or nodular on the surface. Sometimes the indurated masses of tubercular infiltration are felt as bands running across the abdomen; thus, the thickened omentum often forms a transverse band at its upper part, and the tissue about the obliterated urachus forms a vertical band below the umbilicus. In many cases the skin around the umbilicus, for a distance of from one to three or four inches, is reddened and infiltrated, pitting slightly on pressure: this is probably due to obstruction of the veins by the tubercular growth. The percussion resonance is variable, and often muffled. Occasionally the abdomen is retracted from the predominance of fibrous tissue and consequent contraction.

Diagnosis.—Besides the general features of pain, distension and emaciation, the recognition of the tubercular masses and the inflammatory redness about the umbilicus, are the most important. The infiltrated omentum may be mistaken for the lower part of an enlarged liver, but the resonance of the stomach above it should prevent this error. Sometimes, but not always, the diagnosis may be confirmed by the presence of tubercle in other parts of the body. In children or young people, a simple ascites, otherwise unexplained, is likely to be tubercular, but it may be confounded with ascites from cirrhosis of the liver, or even with an ovarian cyst. The liquid withdrawn may be inoculated into animals, but the results are not always conclusive.

Prognosis.—This is more hopeful than the prognosis of many other tubercular lesions. Death results from exhaustion, or from acute tubercular inflammation in the pleura, pericardium, or meninges. But many patients treated early have apparently recovered completely.

Treatment.—This may be conducted on the same principles as that of chronic peritonitis. Mercurial applications should be constantly applied for weeks or months. Cod-liver oil and general tonics should be used internally. The diet should be easily digestible, and stimulants in moderate amount may be given. A considerable number of cases have now been successfully treated by opening the abdomen, washing out or sponging the surface of the peritoneum, and inserting a drainage tube.

NEW GROWTHS IN THE PERITONEUM.

One of the most common growths in the peritoneum is *carcinoma*, secondary to disease in the viscera, especially the stomach and the ovary. It is more common in females than males, and occurs mostly at an advanced age. It occurs in the form of flat circular deposits, covering the abdominal surface, and, like tubercle, it is most abundant on the diaphragm and in the flanks; similarly, the omentum may be thickened and infiltrated, and eventually large cancerous nodules may occur all over the abdomen. In its structure it is generally a true carcinoma, very fibrous, and yielding but little juice. Colloid cancer is present in a certain number of cases. Considerable liquid effusion is commonly present (*cancerous peritonitis*), and blood is not infrequently mixed with it, so that it acquires a brown, brownish-red, or even red colour. Occasionally nodules of cancer are felt in the skin round the umbilicus, and the glands in the groin may be infiltrated with the same growth.

Sarcoma is another common form of malignant disease: it occurs in the retro-peritoneal tissues, in the omentum, mesentery or broad ligament.

Fibroma, lipoma, hydatid, dermoid, and other *cysts* of less certain nature also occur.

Symptoms.—The more rapid and malignant forms of growth are characterised by pain, emaciation, cachexia, and the presence of the tumour; if inflammatory conditions predominate there will be a resemblance to other forms of chronic peritonitis. In other cases the intestine may be involved (*see* Intestinal Obstruction).

Prognosis is absolutely unfavourable, and **Treatment** must be directed to the relief of symptoms, the temporary removal of fluid when it is considerable, or an operation for obstructed bowel.

ABDOMINAL TUMOURS.

It may be well to allude in this place to the great number and variety of tumours which may be found in the abdomen, and the diagnosis of which often presents considerable difficulties. Every one of the contained viscera may give rise to tumour, and to describe all these would only be to repeat what has been already said—or will be said—in the accounts of diseases of special organs; and their differential diagnosis must be gathered from the same source. It will be sufficient here to enumerate the more important tumours, and subsequently to describe one which has not been dealt with elsewhere—namely, aneurysm of the abdominal aorta.

The tumours are as follows:—*Liver:* general enlargements, abscess, cancer, hydatid, distended gall-bladder; *spleen:* enlargements, abscess, cancer, hydatids, infarcts; *stomach:* cancer, dilatation; *intestines:* cancer, lymphoma, strangulation, intussusception, appendicitis, matting by tubercular disease, actinomycosis, fæcal accumulation; *pancreas:* cyst, cancer; *kidneys:* enlargements, moveable kidney, abscess, cancer, hydatid, hydro- and pyonephrosis, tubercular pyelitis; *supra-renal capsule:* cancer, tubercle; *peritoneum:* chronic peritonitis and matting, tubercular peritonitis, localised suppurative peritonitis, cancer, hydatid, and other cysts; *lymph-glands:* tubercle, cancer; *uterus and appendages:* pregnancy, fibro-myoma, cancer, cysts of the broad ligament, hæmatocele, pelvic cellulitis, ovarian tumours and cysts; ***bladder*** : **retention** of urine.

Tumours of the abdomen are also simulated by the **prolapse of** all the organs, liver, spleen, kidneys, stomach and intestine, which occasionally occurs, and is described under the name of ***enteroptosis*** or *Glénard's disease.* This occurs especially in women; and the effects are most noticeable in the erect posture. The lower part of the abdomen is prominent or pendulous, the upper part flattened. It may be unaccompanied by symptoms, or there may be such troubles as nausea, vomiting, pain and distress after food, and various neurotic symptoms. Relief is afforded by wearing a supporting belt; and in extreme cases by operation.

ABDOMINAL ANEURYSM.

Ætiology.—This has much the same causation as aneurysm in other situations. It is common in middle age, and is more frequent in males than in females.

Pathology.—The usual seat is between the diaphragm and the origin of the superior mesenteric artery, and it often involves the origin of the cœliac axis. In its growth it may interfere with adjacent organs, press upon the vena cava, or erode the vertebræ. Aneurysms of the superior mesenteric, or of the iliac arteries, are less common, and will not be specially considered here.

Symptoms.—These are pain, the presence of a pulsating tumour, with murmur, and sometimes evidences of pressure. The *pain* is situate in the abdomen, is often severe, paroxysmal or neuralgic in character, and may radiate to either side, into the groin, or the back. The *tumour* varies, of course, with the seat of the lesion; it is more common in the epigastric region, in the middle line or slightly to the left; it is globular or ovoid, pulsatile, and expansile; it is scarcely, if at all, affected by the movements of the diaphragm. A systolic murmur can be generally heard over it. The pressure signs other than pain are not common, since the various organs readily yield to its progress. But it may exceptionally cause jaundice by obstructing the bile-duct, or dropsy by pressure on the vena cava. It may press on the colon, or on the stomach, and vomiting is sometimes present. The duration may be two or three years, and death results, as a rule, from rupture of the sac into the retro-peritoneal tissue, into the peritoneum, or into one of the hollow viscera; or from the exhaustion of pain, sleeplessness, sickness, and malnutrition.

Diagnosis.—Abdominal aneurysm has to be diagnosed from the *excessive pulsation* of the aorta previously described (p. 573), and from tumours lying in front of the aorta, especially *cancer of the stomach*, to which pulsation is communicated from the healthy aorta. The former is distinguished by the absence of murmur, the normal size and shape of the vessel, and the neurotic or hysterical character of the patient. Tumours over the aorta do not expand laterally, and are often irregular or nodulated in shape; the pulsation in some cases ceases when the patient is placed prone, or on his hands and knees, so that the tumour may fall away from the aorta. Cancer of the stomach is displaced more than an aneurysm by a deep inspiration.

Treatment.—This must follow the lines indicated under the head of Thoracic Aneurysm (p. 568). But an abdominal aneurysm is sometimes open to treatment by proximal or distal compression by the tourniquet, or other surgical means.

DISEASES OF THE DUCTLESS GLANDS, LYMPHATIC SYSTEM, AND BLOOD.

DISEASES OF THE THYROID BODY.

BRONCHOCELE.

(*Goître.*)

By bronchocele or goître is meant hypertrophy of the thyroid gland.

Ætiology.—The causation of goître is still a matter of doubt. The most prominent fact is that it is frequent in certain localities, and especially in limestone or magnesian-limestone districts. In England it occurs in Derbyshire, and to a less extent in Devonshire. On the Continent it is frequent in the mountainous regions of Savoy, Switzerland, Northern Italy, the Tyrol, and Styria. It has thus been generally referred to the influence of the limestone formation upon the water which the inhabitants drink. But there are difficulties in accepting this hypothesis; and while some refer it to other defective hygienic conditions—such as bad ventilation, overcrowding, and filth accumulation—others believe that it must be due to some miasmatic influence, or to an actual micro-organism which is especially developed over certain rock formations. In Central Europe it is often found associated with ***cretinism.***

In England, goître is most frequent in young women.

Morbid Anatomy.—Goître occurs in the following forms:—(1) A simple soft hypertrophy; (2) cystic bronchocele; (3) fibrous bronchocele, and (4) fibro-cystic bronchocele. The enlargement may affect one lobe; or both, or the whole gland; but the isthmus is rarely involved alone. Cysts result from the distension of the normal follicles of the gland, and become filled with a colloid material; the fibrous tissue results from hypertrophy of the connective tissue between the follicles. The size varies from a moderate prominence of the neck on either side, to a mass as large as the fist or a fœtal head, which hangs down in front of the upper part of the sternum.

Symptoms.—Enlargement of the neck and a feeling of fulness are often the only symptoms. If the goître is very large, there may be dysphagia from pressure on the œsophagus, or dyspnœa from compression of the trachea, or of the recurrent laryngeal nerves.

Diagnosis.—This is, as a rule, easy. The thyroid nature of any enlargement is proved by its movement up and down with the larynx during the act of swallowing.

Treatment.—The patient should remove from the locality where the disease is known to be prevalent, and every hygienic condition should be made perfect. Iodine is the drug which has the most influence upon goître. Iodide of potassium, with tincture of iodine, should be given internally, or the vapour of iodine may be constantly inhaled. Externally, the tincture or ointment may be applied. Iron is sometimes useful as well. Woakes recommended fluoric acid in doses of 30 minims three times daily. In any case the treatment may have to be continued for months. Surgical measures may be necessary in very hard or very large goîtres; they are the injection of tincture of iodine, the use of setons, and the removal of the gland by operation.

SPORADIC CRETINISM.

Cretinism is a condition characterised by stunted growth, a large broad head, thick features, wide separation of the eyes, flat nose, large mouth, much wrinkling of the coarse and rough skin even in early life, narrow chest, full abdomen, crooked legs, and considerable mental deficiency, amounting to idiocy. Many *cretins* have enormous goîtres; in others the thyröid is absent. Occasional cases of a similar disease, *sporadic cretinism*, have been observed in England, and some of these have had slight goîtres. Fagge, who described such cases, also noted the occurrence of fatty subcutaneous tumours above each clavicle.

The local agent, whatever it is, which causes goître after a limited exposure, seems, in the course of two or three generations, to produce cretinism.

Treatment.—The close resemblance in many respects between cretinism and myxœdema has led to the employment of the same remedy—namely, thyroid extract (*see* Myxœdema), and with much success. Under its influence children have grown rapidly, have lost the œdematous infiltration of the tissues, and have become more intelligent.

EXOPHTHALMIC GOÎTRE.

(*Graves' Disease. Basedow's Disease.*)

This curious affection was first described by the Dublin physician, Graves, in 1835, and by a German physician, Basedow, in 1840. It consists essentially of three conditions: protrusion of the eyeballs, enlargement of the thyroid gland, and increased action of the heart.

Ætiology.—It occurs much more frequently in women than in men, and mostly between the ages of fifteen and thirty. It has been thought sometimes to follow upon anæmia and chlorosis; but its early symptoms may have been mistaken for one of these. More often there has been a neurotic tendency, as shown in hysteria or epilepsy, or mental disease in the family. In a few cases it has followed rapidly upon some emotional or mental excitement, or even direct injury to the head. A hereditary connection has sometimes been observed; once the disease affected mother and son; more often it attacks two or three brothers or sisters in the same family.

Symptoms.—With the exceptions just alluded to, it generally comes on quite gradually, and, as a rule, the cardiac symptoms first appear, the protrusion of the eyeballs and the thyroid swelling not till some months later. Occasionally they may appear in a different order, or one or other of the three chief symptoms may be absent; but the circulatory trouble is the most constant. In the fully developed disease, the *heart* beats quickly and forcibly; the impulse is in the natural position, but it is felt over an abnormally large area; there is often a systolic murmur in the pulmonary area, and occasionally one at the apex. The carotids and large arteries pulsate with great force, and the patient feels the violence both of the cardiac beat and the arterial throbbings. The pulse reaches 120, 130, or 140 in the minute, is of medium size, and yields no special sphygmographic tracing. The patient suffers from shortness of breath on exertion in proportion to the cardiac disturbance. In course of time the heart may become dilated and hypertrophied.

The enlargement of the *thyroid body* is symmetrical, usually moderate in dimensions, and rarely equal to that of the larger endemic goîtres. If the hand be placed over it, a thrill can be felt which is due to the movement of blood in its dilated vessels, and a systolic murmur can be heard with the stethoscope.

The *prominence of the eyeballs* (exophthalmos, proptosis) is the most striking characteristic of the disease, and gives to the patient an unpleasant terrified appearance. It affects both eyes, and may reach such a degree that the sclerotic is seen both above and below the cornea, and the eyelids are unable to meet. Even when the eyelids can be voluntarily closed, they may lie apart during sleep; and if the exophthalmos is extreme, there may be irritation and ulceration of the cornea as a result of exposure. Sometimes the prominence is only simulated in consequence of spasm or retraction of the upper eyelid (Stellwag's sign). In association with the exophthalmos is another symptom, which the discoverer, von Graefe, regarded as pathognomonic—namely, that when the patient lowers the eyeball to look down, the upper eyelid is not depressed to a corresponding extent—a want of uniformity in the movements of the eyeball and the upper lid. This, however, is not present in every case, although it has sometimes been noticed even before the protrusion. On the other hand, when present it is of importance, as it does not occur in other kinds of exophthalmos. The pupil and accommodation are unaffected; and the ophthalmoscope reveals little beyond overfull and tortuous retinal veins.

The sufferer from these lesions is out of health in many ways besides. She is languid, anæmic, unfit for exertion, wanting or capricious in appetite, subject to nervousness, headache, vertigo, fits of temper or crying, irritable, or hysterical. There is often a more or less constant *tremor* of the limbs, and even of the whole body. The nervousness and cardiac action are increased by attention or by any excitement. The patient is generally thin, and may waste a good deal. A moderate degree of fever is often present; and some patients show various pigmentary disturbances of the skin, such as moderate bronzing, chloasma, or leucodermia. A subjective feeling of heat, flushing of the head and neck, sweating, diarrhœa, glycosuria, and intermittent albuminuria have been noticed in different cases. The electrical resistance of the body is diminished as a result of the moisture of the skin. The voice is often thin, feeble, or piping, sometimes, perhaps, from pressure of the goître on the trachea. The symptoms are liable to aggravation from time to time.

Morbid Anatomy.—The important change in the enlarged thyroid is that the epithelial cells lining the vesicles become cylindrical instead of cubical, that they proliferate into the vesicle, and that the contents of the vesicles lose their colloid nature, and become more mucous and granular. These changes resemble those which take place in compensatory hypertrophy of the remains of the gland after partial removal (Edmunds). In later stages it may become fibrous or cystic.

The thymus gland is often persistent and enlarged, and there is sometimes enlargement of the spleen, of the cervical or bronchial glands, or of Peyer's patches in the intestine.

Pathology.—This is, as yet, very obscure. The protrusion of the eyeballs is probably in part due to venous congestion, and in part to an overgrowth of fat in the orbit. A third factor is believed by some to be present, namely, a contraction of certain non-striated muscular fibres, which have been recognised by Müller, in the membrane lining the orbit over the spheno-maxillary fissure. Such a contraction, it is thought, would force forward the contents of the orbit. The presence of similar smooth muscular fibres in the upper (and lower) lids is considered by von Graefe to explain the symptoms described by him, since they are innervated by the sympathetic. Others ascribe them to weakness of the orbicularis palpebrarum. When it is attempted to explain how these different lesions can be referred to a common origin, considerable difficulty arises. The three cardinal symptoms suggest a lesion of the cervical sympathetic; but nothing has been found in the cervical ganglia or in the brain or spinal cord to explain the symptoms.

The resemblance of the symptoms to those which follow large doses of thyroid extract (p. 727) has suggested the view that this disease is due to an excess of the supposed *internal secretion* of the thyroid; and improvement has occurred in some cases after partial excision of the hypertrophied gland.

Diagnosis. — Occasionally a *simple goître* may actually press upon the sympathetic in the neck, and produce dilatation of the pupil, paralysis of accommodation, slight exophthalmos, and depression of the temperature in the external meatus. It will be seen that these symptoms agree with those of Graves' disease only in the exophthalmos. The difference would be most obvious if they occurred on one side only.

Prognosis.—Many cases are of some years' duration, beginning insidiously, and only slowly recovering; others improve up to a certain point, and then remain stationary; others, again, die from exhaustion, from cardiac dilatation and mitral regurgitation, from intercurrent complaints, or suddenly, and then probably from heart-failure; in a few cases myxœdema has supervened. Of twelve cases collected from the Guy's Hospital records from 1877 to 1884, seven had died by 1886 (Hale White). In two other series of patients who had suffered five years or died, the deaths were 25 per cent., recoveries or great improvement, 60 per cent. (Williamson, Ord and Mackenzie).

Treatment.—A cure can only be expected from prolonged treatment, and no drug can be said to have been uniformly successful. Digitalis and belladonna seem to have done most good, and ergot has been recommended. Iron is of little value. Many cases have benefited under galvanism, the constant current being applied with the kathode on the cervical spine, and the anode over the sympathetic in the neck, or over the thyroid; and a weak current may be similarly applied to the eyes. The application of ice to the thyroid is also of value. Thyroid and thymus extracts have been used with no great success; and various operations have been devised, such as ligature of the arteries, partial removal of the gland, stripping the capsule from the gland and leaving the latter outside the wound to shrink, and division of the cervical sympathetic.

MYXŒDEMA.

The prominent features of myxœdema are a peculiar swelling of the skin and subcutaneous tissue, with dryness and roughness of the surface; pronounced mental failure, consisting of dulness, apathy, hebetude, slowness of speech or action; and atrophy or other destructive change of the thyroid body.

Ætiology.—It is much more frequent in women than in men, and, in the majority of cases, the symptoms begin between the ages of thirty and fifty, though they have begun as early as eight and a half and as late as sixty-seven. Some indications of heredity have been observed, and it has been more often seen amongst the poorer classes; but no influence can be ascribed to occupation, climate, soil, or to the conditions which are associated with endemic bronchocele.

In reference to preceding diseases, the chief conditions that seem to be of importance are the existence of phthisis and neurosis in near relations, and mental disturbance in the patient herself. Nothing can be positively stated about the menses. Some patients have had numerous pregnancies, but in others the symptoms have abated during pregnancy, and relapsed afterwards.

Symptoms.—These are at first insidious, so that in most cases the disease has not been noticed till it has been well developed. The appearance is then characteristic; the face is broader than it was in health, and the nose, eyelids, and lips are thicker; its colour also is markedly yellow with a rather bright red patch on each cheek, and deep red or almost livid lips. The skin of the body generally is thickened, and the legs and feet have the appearance of slight œdema, although in many cases, but not in all, pitting is entirely absent. The shape of the hand also undergoes changes; it becomes broader opposite the heads of the metacarpal bones, and the fingers become thick and uniform in shape; this change has received the not very distinctive name of "spade-like." The feet are similarly affected. Perspiration is often deficient or absent, the skin dry and scaly, and the hair falls out, leaving only a thin covering on the head, or causing actual baldness of the scalp, eyebrows, and eyelids. The nails are stunted and brittle. The mucous membranes show the same change; at any rate, the uvula and soft palate are swollen, and the tongue is large and thick; moreover, the teeth become carious or loose. The nervous system of the patient is the next thing that strikes one: she appears dull, apathetic, slow in thought and movement. She speaks languidly and deliberately, as if the thick tongue mechanically interfered with articulation, but the slow movements of the eyes and the muscles of expression accompanying speech indicate that the neuro-muscular apparatus is also faulty. Articulation is thick or blurred, and the voice is monotonous. The movements of the body and limbs are correspondingly slow. On the mental side, memory is defective, the patient is often irritable, or suspicious, or dull and sleepy; and hallucinations, delusions, and convulsions have been noticed in a certain proportion of cases. The temperature is mostly subnormal, the patient suffers from cold very readily, and the hands and feet are often cold and blue. Examination of the organs, as a rule, gives negative results, at any rate during the greater part of the illness.

The pulse is weak or slow, but high tension has not been often recorded. The bowels are constipated. The urine is of low specific gravity, the urea less than normal, and albumen is mostly absent, though it may appear towards the end of the case. Where the thyroid gland could be felt it has nearly always been described as small; but in the majority of cases there is some difficulty in satisfying oneself of the condition of the organ, or even of its actual presence; but the same, perhaps, may be said of persons who have not got myxœdema. The progress of the case is slow; patients are known to have had the disease ten years or more without material change. Nevertheless, it undoubtedly shortens life; the sufferers die of intercurrent diseases, such as pneumonia, and bronchitis, or sink from general or nervous exhaustion.

Morbid Anatomy.—The number of cases of *post-mortem* examinations of myxœdema that have been recorded is not as yet large, but a certain uniformity of result exists. The changes in the skin are some nuclear proliferation, and development of connective-tissue in the neighbourhood of the sweat-glands, sebaceous glands or hair-follicles. Gelatinous and œdematous skin have only a few times been recorded, and the considerable amount of mucin discovered in one of Ord's early cases (for which the name *myxœdema* was given) has not as yet been found in others. There is a fair amount of subcutaneous fat.

Interstitial nephritis and hypertrophy of the heart were found in a third of the cases recorded in the Clinical Society's Report

(1888), and no marked or constant change in other organs except the thyroid body. This was in every case smaller than normal, pale, yellowish white or buff-coloured, tough or indurated, fibrous, or structureless. The disease appears to begin as a small-celled infiltration of the walls of the vesicles, and epithelial proliferation within them. Later on the gland consists mainly of fibrous tissue, with scattered groups of cells, the remains of the vesicles; and, finally, nothing but dense fibrous tissue is left.

Pathology.—That disease of the thyroid body is the cause of the whole condition of myxœdema is shown by the pathological results, by the results of the removal of the thyroid body in animals, and of operations upon this organ in man (*operative myxœdema*), by the effects of recent methods of treatment, and by the resemblance of the disease to endemic and sporadic cretinism. The explanation appears to be that the thyroid normally supplies an *internal secretion*, requisite for healthy metabolism, which is checked by its atrophy.

Diagnosis.—When once the typical features have been recognised, the diagnosis of subsequent cases is generally easy. No doubt some years ago many cases were regarded as *Bright's* disease with an unaccountable absence of albumen. But myxœdema differs from Bright's disease not only in the absence of albumen, but in the absence of pitting, in the peculiar yellow and red colour of the face, and in the defective mental or neuro-muscular condition. The colour of the face is sometimes even suggestive of *mitral disease;* but the swelling is more than is common in cardiac affections, there is rarely a murmur, and the nervous condition is again distinctive.

Treatment.—Very striking improvement in the condition of patients with myxœdema has been obtained by supplying from animal sources the deficiency of the thyroid body. This has been done in three ways—by grafting a sheep's thyroid in the subcutaneous tissue, as suggested by Horsley; by subcutaneous injection of a glycerine extract of sheep's thyroid (Murray); and by administering the pounded or minced thyroid or its extract internally (H. Mackenzie, Fox). The last is the most convenient method, and thyroid extract can now be obtained in the form of tabloids, one or more of which may be taken daily. The beneficial effects may be seen in the quickening of the pulse, the rise of temperature, the growth of the hair, the diminution of the swelling of the face and hands, and greater mental and vital energy. During this treatment also the skin desquamates over the body. The injections or feedings must be repeated indefinitely to secure a permanent result.

If the thyroid extract or tabloids be given in excess, the patient suffers from headaches, palpitation, quick pulse, muscular tremors, pains in the limbs, thirst and feverishness, much depression and nausea, vomiting or diarrhœa. The occurrence of these symptoms must be the signal to diminish the dose.

Jaborandi (one drachm of tincture three times daily) and pilocarpine have been given with some benefit to increase the perspiration; and the patient is always better when protected from cold.

DISEASES OF THE SPLEEN.

The spleen lies in the upper part of the abdomen on the left side, and is entirely concealed by the ribs. In health its position and size can only be estimated by percussion. There is dulness in the left infra-axillary region over the ninth, tenth, and eleventh ribs, and the included spaces. In front this dulness is limited by a line drawn from the left nipple to the tip of the eleventh rib; behind it reaches nearly to a line continuous with the anterior margin of the latissimus dorsi. If the spleen becomes enlarged, it extends downwards and forwards, the dulness passes in front of the line above mentioned, and if the fingers be placed under the ninth and tenth costal cartilages, while the patient takes a deep breath, the margin of the spleen can be felt to impinge against them. With greater enlargement, it comes distinctly below the ribs at this point, so that it can be readily felt, and occupies more or less of the left upper quarter of the abdomen. In extreme cases of leuchæmia the spleen reaches down to Poupart's ligament, and crosses the middle line below the umbilicus. though it may remain on the left side above. The surface of an enlarged spleen is always dull, is continuous with the lower ribs, and is never overlaid by bowel or stomach. It descends along, and clings to, the abdominal walls throughout. The anterior margin is irregular, and presents one or two distinct notches.

The majority of the disorders of the spleen are secondary to other lesions elsewhere, and I have had occasion to refer frequently to its implication in different forms of fever and in disease of the heart. With some exceptions (leuchæmia, splenic anæmia, and hydatid) the lesion of the spleen is not generally a source of much trouble to the patient. The chief symptom which results from disorder of the spleen is *pain*, which may be present from the formation of infarcts, and from the resulting perisplenitis or abscess, but is not a marked feature in the enlargements which accompany fevers. There may be also a sense of weight from the great size the organ attains in certain cases, especially in leuchæmia. The pathological changes, to which the spleen is liable, and the general symptoms which accompany them, will now be briefly described.

Active Congestion.—The spleen is enlarged in many acute infectious processes, and this is most prominently the case in enteric fever, in relapsing fever, in ague and other malarial fevers, in pyæmia, malignant endocarditis, and acute tuberculosis, and less so in puerperal fever, erysipelas, and syphilis. The capillaries and veins are distended with blood. The splenic pulp is swollen, and the capsule of the organ is distended. After death the spleen is found to be of dark-red or purple colour, and very soft; and the pulp is readily washed away by a current of water.

Splenitis and Perisplenitis.—In some of these infective conditions the process goes beyond the stage of hyperæmia into one of acute inflammation, as shown, according to Ziegler, by the excessive quantity of white cells found within the vessels and pulp. Accompanying the splenitis there may be inflammation of the capsule, or perisplenitis, with resulting adhesions to adjacent organs, or to the abdominal parietes. Abscess is a very rare result of general splenitis. It more often results from the breaking down of infarcts in malignant endocarditis (*see* below).

Chronic Enlargement.—Splenitis may subside entirely, or go on to hyperplastic changes in the pulp, trabeculæ, vessels or capsule. The *ague-cake*, already mentioned (p. 91), is the best example of this. Some enlargement is also seen in rickets, tuberculosis, and Hodgkin's disease (*see* Lymphadenoma). The greatest size is reached in splenic anæmia (*see* below) and in leuchæmia, in which disease the spleen often occupies more than half the surface of the abdomen (*see* Leuchæmia).

Splenic Anæmia.—Rare cases occur in which great enlargement of the spleen is associated with profound anæmia, but without leucocytosis. The first symptoms are either a general anæmia, or some complaint of pain in the left side, probably due to attacks of perisplenitis. The anæmia becomes intense, the red corpuscles may be reduced to one-fourth of their number, and the amount of hæmoglobin is still more diminished—that is, the corpuscles contain less than their normal proportion of hæmoglobin (chlorotic type). The red cells are smaller than normal, and microcytes are present; but there are few poikilocytes, and the leucocytes are unaffected in number and quality. The liver is also slightly enlarged, and there are digestive troubles. Epistaxis is the only frequent form of hæmorrhage until the last stages. Ultimately, in from two to four years, death takes place after progressively increasing anæmia and emaciation. The spleen is found to be greatly enlarged, weighing from two to four pounds, firm, and generally smooth, presenting hæmorrhages and infarcts on section, and showing under the microscope a great increase of the fibrous trabeculæ, with atrophy of the Malpighian corpuscles, and disappearance of the pulp.

Though the cases are rare it is important to recognise them, first, because they may be mistaken for leuchæmia if the blood is not examined under the microscope, and secondly, because the removal of the spleen has been safely and successfully undertaken in some of these cases, whereas it is fatal in leuchæmia. The medicinal treatment should be that of leuchæmia or pernicious anæmia, but is of little use. The disease has been called also *primary splenomegaly*, but the relation of the hypertrophy to the anæmia has not been proved.

Passive Congestion.—The usual causes of venous congestion of the spleen are mitral valvular disease and cirrhosis of the liver; in both the return of blood through the splenic vein is directly impeded. In cirrhosis of the liver, the spleen may be greatly enlarged; in old heart disease it is often small and very hard.

Embolic Infarcts.—These are the results of the impaction of fibrinous particles, detached from the valves of the heart or from thrombi in its cavities. The infarcts form wedged-shaped or conical masses, which may reach a large size, and occupy one-half or two-thirds of the organ. They go through the changes of colour elsewhere described (p. 571), and in septic cases, such as pyæmia and malignant endocarditis, they become purulent, from the presence of bacteria. Infarcts also occur in leuchæmic spleens.

Lardaceous Degeneration.—This change is similar to that which is seen in the liver and kidneys. It affects the Malpighian follicles, the trabeculæ, and vessels. When the Malpighian follicles are alone prominently affected, they appear as grey specks upon the surface (*sago-spleen*); in other cases the organ is more uniformly pale, very hard and enlarged. The diseased parts are coloured brown-red by the addition of tincture of iodine. The lardaceous spleen is enlarged, hard and smooth; the liver and kidneys are often affected at the same time.

Tumours of the Spleen.—*Benign* tumours and *primary carcinoma* are exceedingly rare. *Secondary carcinoma* occurs now and then in scattered deposits, and the *lymphomata* are found in Hodgkin's disease. *Tubercle* appears in the spleen as a part of general tuberculosis, in the form of grey, or often bright yellow nodules, which may reach the size of small peas, scattered throughout the substance and on the surface. *Syphilitic* gummata are rare; but the spleen is sometimes enlarged in congenital syphilis.

Parasites.—Exceptionally a hydatid cyst develops in the spleen and forms a large tumour.

The **Treatment** of splenic lesions is dealt with under the various diseases with which they are associated.

DISEASES OF THE SUPRA-RENAL CAPSULES.

ADDISON'S DISEASE.

(*Melasma supra-renale.*)

This disease was first described by Dr. Thomas Addison in 1855, and presents three main clinical features: namely, (1) great debility, with small feeble pulse; (2) vomiting; (3) a peculiar pigmentation of the skin. In the large majority, if not in all cases, the supra-renal capsules or adrenals are found to have undergone a tuberculous, caseous, or calcareous degeneration.

Ætiology.—Most cases occur in early adult life, or middle age, though young children and elderly persons are not exempt; and it affects males more frequently than females. It appears to be a good deal more common amongst the poor and labouring classes than amongst the well-to-do. As to its immediate causation, it must be noted that in a certain number of cases it occurs in connection with phthisis, or other tubercular disease; in other cases it seems to follow upon inflammatory lesions in parts adjacent to the capsules—for instance, caries of the dorsal or lumbar vertebræ, psoas abscess, or other suppuration in this neighbourhood; in others again it has followed upon injury to the back or loins, without the intermediate occurrence of any local lesion. But in the majority of instances there is absolutely no clue to the origin of the disease.

Symptoms.—The onset is generally insidious, and the patient gradually suffers from weakness, depression, languor, and indisposition for exertion. There may be pains in the loins, hypochondria, or epigastrium, and tenderness on pressure in one or other hypochondriac region. The heart's action is very weak, the pulse small, feeble, and compressible, and there are faintness and giddiness on rising in bed, or breathlessness and palpitation on exertion. Appetite is generally deficient, and nausea, retching, and vomiting are important features of the disease. At the same time, though the patients may not be robust, there is seldom any great degree of emaciation, and a considerable layer of subcutaneous fat may persist to the end. A peculiar *discoloration of the skin* is the symptom which has attracted most attention, but which it is important to remember may be entirely absent. This symptom may be noticed coincidently with the above general symptoms, it may develop occasionally before them, or it may occur several months after they are pronounced. It is presumable, in this last class of cases, that if the general symptoms were very severe they might be fatal before the skin was affected; and thus the occasional absence of pigmentation in Addison's disease of the supra-renal capsules is explained. The pigmentation, or *bronzing*, is, in its lighter shades, dusky, or yellowish-brown; sometimes of olive or green-brown hue. In its more pronounced form the skin has a rich brown colour, like that of a mulatto. The pigment is not scattered or in spots, but more uniformly distributed over different parts of the body—that is to say, large areas are discoloured, the darker tints gradually shading off into the lighter, or into the natural colour of the skin. The change usually invades, first, the parts of the skin which are naturally exposed, such as the face, neck, and the backs of the hands, but not the scalp or the lip under the moustache; secondly, parts which are naturally more deeply pigmented than others, such as the axillæ, penis, scrotum, and areolæ of the nipples; and thirdly, seats of pressure or slight injury, such as the marks of garters and waistbands in women, and places where blisters and plasters have been applied. But the scars of wounds destroying the skin remain white, and are bordered by a deep layer of pigment. On the darkened parts of the skin may be seen small black specks, like moles or freckles. In advanced conditions the whole body may be covered by the pigmentation; but, as a rule, one must be prepared to diagnose the disease, and, indeed, many patients die, before this stage is reached. The pigmentation is not limited to the skin. A bluish-black line may often be seen on the inner side of each lip running along the mucous membrane, parallel to the line of junction with the skin; and other more irregular patches may occur on the mucous membrane of the cheek and on the side of the tongue. Some of these may be determined by the presence and consequent irritation of carious teeth. The temperature is, as a rule, normal, the urine of medium colour and specific gravity, and free from albumen.

The course of the disease is very variable. It is often marked by exacerbations and remissions, periods of severe illness, which confine the patient to bed, alternating with times of comparative health; but after each fresh aggravation of the disease the patient is left decidedly worse than he was before it. The duration varies from a few months to six or seven years. Death takes place mostly by asthenia, the patient getting gradually weaker, and lapsing into a drowsy or semi-comatose condition, with increasing feebleness of pulse. Delirium and convulsions occasionally close the scene. In some cases the general symptoms and a very slight pigmentation have been noticed only for some months, when extreme prostration has ensued and carried off the patient in a few weeks.

Anatomical Conditions.—The parts of the body presenting changes are the skin and mucous membrane, the supra-renal capsules and the adjacent parts, and the mucous membrane of the alimentary canal. The change of colour in the former is due to pigment, which is deposited, for the most part, in the deepest layers of the epidermis, a condition similar to that which obtains naturally in the skin of the negro. A few pigment-granules are also scattered in the upper parts of the papillary layer of the true skin.

The supra-renal capsules are usually enlarged, hard, and irregular, and present on section a combination of translucent greyish or greenish-grey tissue, and opaque yellow cheesy substance. There are also conditions intermediate between the two; or the cheesy substance may exist only in the form of nodules in the midst of the other. Small particles, or larger masses of cretaceous matter may be found in the caseous substance; or the whole gland may be converted into a cretaceous mass. Sometimes, on the other hand, the caseous matter has softened down into a cavity containing pus. It is obvious that this condition has the closest possible resemblance to a tubercular process, and the identity has been recently shown by the discovery in a few cases of the tubercle-bacillus. But another important feature must be noticed—namely, that the chronic inflammation of the supra-renal body leads to thickening of the connective tissue around it, and adhesion to neighbouring structures; and that the solar plexus, the semilunar ganglion, and the terminations of the phrenic and pneumogastric nerves are often involved in the disease either by compression or acute inflammation. Enlarged lymphatic glands are also found in the surrounding connective tissue, as well as in the mesentery, and behind the peritoneum. The solitary follicles of the small and large intestines are often swollen, and the mucous membrane of the stomach may be "mammillated" from an overgrowth of lymphoid tissue between the gastric tubules.

In some cases there are found the tubercular changes in the lungs or other parts, or the various abscesses, to which allusion was made when treating of the ætiology of the disease.

In a small number of instances, in which the symptoms of Addison's disease seem to have been unequivocally present, the capsules have been atrophied to an extreme degree, without evidence of previous tuberculisation.

Pathology.—The nature of the disease is still under discussion. Since the vast majority of cases show tuberculisation of the adrenals, and since, on the other hand, these bodies may be transformed into masses of cancer without producing the symptoms of Addison's disease, it has been held by many that the former process is the only true Addison's disease, and that the symptoms are due not to destruction of the adrenals, but to the implication of the important nervous structures in their neighbourhood. This view appears to receive confirmation from the occasional occurrence of pigmentation of the skin when these nerves and ganglia are involved in lymphadenomatous disease. But the cases of simple atrophy localise the lesion in the adrenals themselves. The explanation then turns upon our view of the function of the gland. If it normally separates useless pigment and proteids from the blood, as MacMunn suggests, the pigmentation and toxic condition would be accounted for by its destruction; but the theory remains unproved. The alternative view is that the adrenals, like the thyroid gland, secrete a substance which is essential to health, and which is deficient as a result of supra-renal disease; and that thereupon the body suffers in some way yet to be explained.

Diagnosis.—The mistakes most likely to be made are: (1) to take some other discoloration for that of Addison's disease; (2) to fail in recognising the symptoms when the pigmentation is slight or absent. The discolorations likely to be mistaken for it are slight *jaundice*, which is distinguished by the yellow tinge of the conjunctiva; *phtheiriasis*, to be recognised by the scars, blood-crusts, scratches, pediculi, the limitation of the colour to parts that can be scratched, and the entire freedom of the face; the sallow or earthy tints of *malaria* and of *phthisis*; *chloasma uterinum* in women; and *tinea versicolor*. In early stages, without much darkening, the apparently causeless weakness, with small feeble pulse and sickness, are the diagnostic features.

Treatment.—This must be, on the whole, tonic. Sickness must be met by effervescing salines, bismuth, iodine, &c. Iron and strychnine are the most suitable tonics. Dr. Greenhow thought cod-liver oil did not agree, and preferred glycerine. The internal administration of a supra-renal extract is justifiable; but the results at present are not very satisfactory.

DISEASES OF THE LYMPHATIC SYSTEM.

The majority of the diseases to which the lymphatic system is liable arise as the result of the passage into the lymph-vessels of some substance foreign to them, such as micro-organisms, tumour-cells, or other solid particles, and the poisons of certain diseases which may ultimately prove to be of microbic nature. These either set up acute inflammation or cause a change in the gland of the same nature as the source from which the foreign substance has come. Thus we see the inguinal lymph-glands inflamed in syphilis, the glands of the jaw in diphtheria; the axillary glands in poisoned wounds of the arm; the bronchial glands in pneumonia; and the mesenteric glands in enteric fever. On the other hand, the bronchial glands become tuberculous as the result of phthisis; and the axillary glands are cancerous in consequence of carcinoma of the breast. Inflammation of the lymph-glands either goes on to suppuration, or subsides as the primary cause is removed, or becomes a chronic induration. Tubercle and cancer run the same course as they do in other parts. Hodgkin's disease or lymphadenoma, and tuberculosis of the mesenteric glands will be separately described. The inflammation and suppuration of the cervical glands, which is often, but not always, a tubercular process, and probably depends on infection from the tonsils, are generally dealt with in surgical works.

The lymph-vessels are inflamed as a result of septic poisons, as for instance in lymphangeitis, or absorbent inflammation, which involves the lymphatics between a wound and the nearest gland. Obstruction of lymph-vessels occurs under certain circumstances in tropical climates, especially by the nematode worms, *filariæ*. These conditions will be described under the name *Filariasis.*

HODGKIN'S DISEASE.

(*Lymphadenoma.*)

Dr. Hodgkin first described, in 1832, a series of cases of enlargement of the lymphatic glands with a peculiar deposit in the spleen; and similar cases have been recorded by other writers as progressive multiple hypertrophy of the lymph-glands (Wunderlich), multiple malignant lymphoma (Billroth), and adénie (Trousseau). The name *lymphadenoma*, indicating the nature of the new growths which occur in the disease, is now mostly used in England, while German writers employ the term *pseudo-leuchæmia*, from its apparent relations to leuchæmia. It has also been called *anæmia lymphatica.*

Ætiology.—Very little is known of its causation. It occurs at all ages, but is most frequent in early and late adult life. Men are more often attacked than women. In a few cases depressing causes, such as intemperance, insufficient food, exposure to cold, and parturition have been noted; and in a good many instances the disease seems to have been brought about by a local lesion, such as a blow, a discharge from the ear, an abscess or an eczema, in consequence of which a group of glands is irritated and enlarges, and subsequently the other glands in the body are involved. More often, however, no cause whatever can be discovered.

Symptoms.—The chief clinical features of the disease are enlargement of the lymphatic glands and anæmia. Generally, the *lymphatic enlargement* occurs first, the change beginning in the cervical glands in most cases, and subsequently involving those of the axilla and the groin. The glands form irregular and nodulated masses of different sizes, extending, perhaps, from the clavicle to the jaw, or in the axilla of such a size as to prevent the arm being applied to the side. The individual glands are as large as a pigeon's egg or a hen's egg, are either soft or firm, usually painless, and at first freely moveable under the skin upon one another. Subsequently they may become adherent, but rarely caseate or suppurate. Other smaller groups of glands are also involved, such as the occipital; and the change affects the glands in the interior of the body—namely, the retro-peritoneal, bronchial, mediastinal, and mesenteric. In many of these regions the growth of the glands may be such as to cause serious pressure on the neighbouring parts. Thus in the neck, the larynx, trachea, and œsophagus; in the thorax, the large veins, and the recurrent nerves; in the abdomen, the nerves of the solar plexus; in the axillæ and groins, the nerves supplying the limbs are liable to compression, producing the characteristic symptoms. The implication of the solar plexus may cause pigmentation of the skin.

The *spleen* is, as a rule, only moderately enlarged; it projects a little below the left costal margin, or occupies the left upper quarter of the abdomen; but if it attains the same size as that seen in splenic leuchæmia, the case is probably not a simple lymphadenoma (*see* Diagnosis).

Anæmia is a prominent symptom, even comparatively early, in Hodgkin's disease. The red corpuscles are diminished to 60, 50, or 40 per cent. of the normal; the hæmoglobin is diminished to a still greater extent (chlorotic type); and in severe cases poikilocytes and nucleated red cells (normoblasts) are seen. The leucocytes are generally not increased in number, but when the glands are soft (or inflamed) there may be a moderate degree of leucocytosis. The urine is only occasionally albuminous. The temperature is often raised, and is either continuously above normal, or shows daily remissions, or periods of fever of two or three weeks' duration, alternating with similar periods of apyrexia.

A certain amount of weakness is soon observed, and as the disease progresses the effects of the anæmia become more pronounced. There is generally a good deal of dyspnœa, partly from the anæmia, and partly from mechanical interference with the trachea, bronchial tubes, or lungs. In time, also, œdema of the lower extremities takes place, with, perhaps, ascites, pericardial effusion, or hydrothorax; and hæmorrhage from the nose or gums, or under the skin, may occur as in other severe blood-lesions. Finally, death is caused by exhaustion, suffocation, or starvation from the pressure of enlarged glands, by hæmorrhage, by cerebral disturbance, coma or convulsions, by pneumonia, pleurisy, or œdema of the lungs. The duration is generally several months, or one or two years; occasionally the cases are much more rapid.

Anatomical Changes.—On *post-mortem* examination, it is found that there are two important changes: one, the enlargement of the ordinary lymph-glands; the other, the growth of tumours having a similar (lymphadenomatous) structure, in different organs of the body, and in subserous and other tissues, probably in most cases arising from natural lymphatic formations. The consistence of the glands varies in different cases of lymphadenoma. They are light grey, or greyish-white on section, and the softer kinds yield, on scraping, a turbid juice, consisting mainly of leucocytes. Microscopic examination shows that the enlarged gland consists of lymphoid corpuscles in a nucleated reticulum; and in the harder varieties this reticulum is abundant, and the corpuscles relatively less, while in the softer varieties the corpuscles are abundant, and the reticulum is very fine. Adhesion of the glands to one another is caused by the lymphoid corpuscles invading the capsule of the gland, and infiltrating the interglandular tissue. Occasionally, as already stated, a little caseous change may occur in a few glands.

The lymphadenoid growths that are independent of the glands proper occur in the spleen, kidneys, liver, lungs, and other parts. The spleen is generally larger than normal, and may weigh even 30 ounces; it is moderately firm, and presents on section a number of white or yellowish tumours, from one-eighth to half an inch in diameter, scattered through its substance, giving it an appearance which has been compared, not inaptly, to "hard-bake." These tumours arise from the Malpighian corpuscles, and consist of lymphoid corpuscles in a reticulum. Similar tumours occur scattered through the liver or the kidneys, which may also show a more diffuse growth of lymphoid tissue; and swelling due to similar growth may occur in the tonsils, and in the follicles of the pharynx, stomach, and intestine. Nodules of growth are also present in the lungs, and soft, pinkish-grey, flat masses, having a similar structure, have been found under the pleura and other serous membranes. In one case under my care, growths the size of peas had formed in pleural adhesions, which stretched across the fluid of a hydrothorax. The epididymis and testicle have been invaded. Sometimes in this disease, also, the bone-marrow is affected, being converted into a reddish-grey semi-diffluent matter.

Pathology.—This is still very obscure; but the resemblance to the spread of malignant growths will not fail to be noticed, especially in those cases where an irritation has caused the enlargement of one set of glands, and others have been affected subsequently.

Diagnosis.—It may be difficult to distinguish the early stage of Hodgkin's disease of the glands from tubercular enlargement, especially when the growth is confined to one set of glands. *Tubercular changes* affect more often a single group, occur in young people, and lead to caseation and suppuration. *Cancer* of the glands may resemble Hodgkin's disease, but secondary deposits are not so likely to take place in other glands as in the different viscera. If the blood contains an excess of white corpuscles, the classification of the case must depend on the relative numbers of the different leucocytes present (*see* Leuchæmia).

Treatment.—Arsenic has been of great benefit in some cases: it should be given in increasing doses until as much as 15 minims of the liquor arsenicalis three times daily is being taken. Phosphorus has also been thought to do good, but this is less certain. The more usual tonics—iron, cod-liver oil, quinine, and others—have no influence. In cases where the enlargement has been confined for some time to one set of glands, their excision has delayed the disease, or even apparently cured it entirely. But this, according to Gowers, is not to be recommended where the anæmia is such that the red corpuscles are below 60 per cent. of the normal.

TUBERCULOSIS OF THE MESENTERIC GLANDS.

(*Tabes mesenterica.*)

Ætiology and Pathology.—The mesenteric glands undergo the same changes of tuberculisation, caseation, calcification, and occasionally suppuration, as are seen in the bronchial glands. Similarly the process may be secondary to a tubercular lesion of the intestine—namely, the tubercular ulcer, or it may be associated with tubercular peritonitis; but it is not infrequently, especially in young children, a primary lesion. The glands are increased in size, and form large masses, which may become perceptible on examination of the abdomen. As a secondary result they cause chronic and tubercular peritonitis; and in the event of suppuration, the abscess may burst into the peritoneum, and set up a general acute inflammation. The disease is much more frequent in children than in adults, and there is a possibility that it is caused sometimes by the ingestion of milk infected with tubercle.

Symptoms.—When we consider that the mesenteric lymph-glands are those in connection with the lacteals, it will be readily understood that any extensive disease in them will seriously interfere with nutrition. Patients with mesenteric disease are wasted; and the small arms, legs, and chest contrast strikingly with the abdomen, which is large, partly from the increase in size of the glands, and partly from the distension of the intestines by flatus. The enlarged abdomen is resonant, and the distended bowels mostly prevent the glands from being felt. Sometimes, however, they are of sufficient size to be recognised by the hand. The accompanying symptoms are diarrhœa with griping pain, and febrile action with malaise. The motions are brown, watery, and offensive. If tubercular ulceration is also present the diarrhœa is likely to be more constant, and the motions are sometimes lighter brown or yellow. Peritonitis is indicated by more pain, tenderness, and abdominal tension.

Diagnosis.—There are three conditions in children, characterised by wasted limbs and a large abdomen, which are liable to be spoken of as mesenteric disease, and "consumption of the bowels." They are tabes mesenterica, tubercular peritonitis, and simple indigestion with diarrhœa. Of these the last is the most common. The diagnosis of tabes can rarely be decided upon unless the enlarged glands are felt, and this is usually prevented by the inflated bowels. Their existence may be suspected if there is continued febrile action, and a tubercular family history; and if the symptoms persist in spite of such changes in the diet as would probably cure simple indigestion or diarrhœa.

Prognosis.—This is not necessarily unfavourable, so long as the change in the glands is not complicated with ulceration of the bowel, or tubercle elsewhere, or extensive chronic peritonitis.

Treatment.—The implication of the lacteals has suggested that the food should not contain too much of a fatty nature. Thus, animal food should be given more abundantly, and what milk is used should be boiled or sterilised. The effect upon the diarrhœa must, of course, be carefully watched; and this may be met with astringents, such as aromatic chalk powder, catechu, Dover's powder, or dilute sulphuric acid. The same tonic remedies should be given from time to time as in bronchial phthisis; but cod-liver oil is not so useful here, on account both of its fatty nature and of the diarrhœa when present. Chloride of calcium may be tried in 5-grain doses, given in milk.

FILARIASIS.

Nine or ten distinct species of the nematode worms *Filariæ* occur as parasites in the human body, of which some infest the connective tissues, and others the blood-vessels and lymphatics. The most important is that of which the embryonal form was first discovered in the blood in 1872 by Lewis and was named by him *Filaria sanguinis hominis*. In view of the four or five other species found by him in the blood, and of the peculiar habit to be mentioned directly, Manson proposes to substitute for it the name *Filaria nocturna*. The different varieties of filariæ occur extensively in tropical and subtropical countries, especially in West Africa, India, the West Indies, and the South Pacific Islands.

Life History of Filaria nocturna.—The parent worm was first discovered by Bancroft, and was called after him *Filaria Bancrofti*. The female is filiform, from 3 to 3½ inches long, and $\frac{1}{100}$ to $\frac{1}{90}$ inch in thickness; the male is much smaller. They have been seen in dilated lymphatic vessels, and there the female discharges an innumerable progeny of embryos, which are sufficiently minute to pass through the lymphatic channels into the thoracic duct, and thence into the blood-vessels, where they are found during the life of the host in extraordinary numbers. These embryos, which were discovered before the parent worm, are also filiform, about $\frac{1}{90}$ inch long and $\frac{1}{3200}$ inch broad; they are contained in a soft covering, in which they elongate and shorten themselves, and which commonly projects for some little distance beyond the caudal extremity. This covering is conjectured to be the shell of the ovum which has been stretched by the growth of the embryo. The embryos are extremely active, twisting and wriggling amongst the blood-corpuscles as they are seen on the slide of the microscope.

In the case of the *F. nocturna*, these embryo filariæ can only be found in the blood-vessels of the skin during the night, from 6 or 7 P.M. to 8 or 9 A.M., whereas during the daytime they are in very small numbers or entirely absent. It is conjectured that they must during the daytime occupy the blood of the deeper vessels; and it has been shown that this diurnal variation depends on the habit of the host. S. Mackenzie, by getting a sufferer from this disease to sleep by day, and eat and walk about during the night, was able to reverse the conditions, finding the embryos present by day and absent during the night hours. In the tropics, the presence of these embryos in the cutaneous vessels at night-time is of importance to their further development, as, like so many other entozoa, they require an "intermediate host" between the stage of embryo and that of complete development. Such an intermediate host is the mosquito, which, while sucking the blood of its human victim, draws up the filariæ into its stomach. Within a few hours of their ingestion, the embryos escape from their sheaths, and pass from the stomach of the mosquito to the thoracic muscles; and here they both develop their organs and grow to a considerable size during the period of six or seven days which are required for the mosquito to deposit her ova on the surface of the water and die. Thereupon it seems that the filariæ, now about one-sixteenth of an inch in length, get into the water and are able to infect human beings by being swallowed when the water is drunk. Ultimately the filariæ establish themselves in the lymphatic vessels of their host, become sexually mature, and furnish the embryos which are found in the blood.

Pathological Relations.—The embryo filariæ may be found in the blood of persons who are in perfect health, and no symptoms appear to attach to their presence in this liquid. They may be readily seen with a low power of the microscope. A film of the blood may be spread on a microscope slide, and examined at once or allowed to dry. Manson recommends that the slide should be dipped in a solution of fuchsin (three or four drops of saturated alcoholic solution to an ounce of water) without previous fixing, and examined without cover-glass.

It is the presence of the adult parasites in the larger lymphatic vessels which gives rise to definite pathological conditions; and this is as a result either of blocking of the channels by masses of the parasite, or more often by inflammatory changes in the walls of the vessels (*lymphangeitis*), which their presence excites.

Thus are produced obstruction of the thoracic duct or other lymph-vessels, and in consequence varicosity of more remote vessels, and the solid œdematous condition of which *elephantiasis* is probably an example. The obstruction to which the thoracic duct may be subject was well shown in Mackenzie's case. He found a large mass of dilated lymph sinuses and glands, extending from the bifurcation of the aorta to the diaphragm, and occupying all the space between the two kidneys. The lower part of the thoracic duct was sinuous and pouched; and above the diaphragm it became impervious, and was lost in tough, dense material, apparently of inflammatory origin. If the thoracic duct is obstructed, the chyle and lymph-vessels below it become dilated and varicose, and the chyle, unable to pass by its usual course, will go along new channels, and thus come into relation with such parts as the walls of the bladder, the tunica vaginalis, or the pleura. By the rupture of dilated lymph-vessels in connection with these parts, chyle or a chylous fluid is extravasated into the hollow cavities, and thus is explained the occurrence of *chyluria*, chylous hydrocele or *chylocele*, *chylous pleurisy*, and *chylous ascites*.

The connection of elephantiasis with filariæ is not so certainly shown, for the blood in these patients is generally free from embryo filariæ; but elephantiasis occurs in precisely the same parts as filarial disease. It is quite clear that elephantiasis is due to lymphatic obstruction, and Manson thinks that from time to time the parent worm, perhaps from injury, gives off prematurely the ova, which are shorter, thicker, and more rigid bodies than the ensheathed embryos hitherto described, because the coverings have not yet yielded to the active movements or growth of the contained embryo; and that such broad ova, which may in course of time be numerous, obstruct the lymphatic channels sufficiently to produce these results, and also by accumulating in the glands prevent the passage of embryo filariæ into the blood-current.

Some of the more important results of filarial obstruction of the lymphatic vessels will now be described.

Chyluria.—The urine is opaque, whitish, or milky in appearance, and has an odour of milk. On standing, a layer of fat may collect on the surface; and generally, also, a soft coagulum forms, which is either transparent or opaque. If it is skaken up with ether, and placed under the microscope, the turbidity is seen to be due to minute oil globules and granules. It contains also a small quantity of albumen. Sometimes blood is present, and gives the urine a pink or darker red colour. Careful microscopical examination of the sediment will often detect the embryo filariæ, which have been already described as being present in the blood, and which have obviously escaped with the lymph or chyle into the urinary passage.

Chyluria, when once established, is not constant; it may disappear, and again recur. When present, it is generally more marked after a meal.

There is not necessarily any disturbance of the health; the patient may seem perfectly well. On the other hand, there is often some uneasiness or pain in the back, loins, or perinæum; and it may be febrile symptoms, debility, or mental depression. Retention of urine, from blocking of the urethra by fibrinous coagula, is sometimes the first sign. In prolonged cases—and it may last twenty or thirty years—there are emaciation, craving appetite, and severe thirst. The disease occurs at all ages, and equally in males and in females.

Diagnosis.—Chyluria is readily recognised, and if it occur in persons from the tropics, the filariæ may be looked for in the blood and the urine. But chyluria and the other lymphatic lesions are occasionally seen in persons who have never resided abroad, and may be exceptionally due to obstruction or rupture of lymphatic vessels produced in other ways. For instance, Whitla records a case of chylous ascites from invasion of the thoracic duct by tubercle.

Treatment.—When once established, little can be done beyond supporting the patient's strength as much as possible, so as to meet the drain of fat, fibrin, and albumen through the urine.

Manson relies on rest in bed, elevation of the pelvis, restriction of food and drink, and gentle purgation, for at least temporary relief.

Lymph-Scrotum.—In *lymph-scrotum*, numerous clear vesicles appear on the scrotum, which is enlarged, soft, and spongy; the vesicles may give way and discharge a fluid, which is obviously lymph. The inguinal glands are enlarged, and there is a liability to the erysipelatoid attacks or fever which occur in elephantiasis. Embryo filariæ may be found both in the blood and in the lymph from the vesicles.

Elephantiasis.—The legs and the scrotum are the parts most commonly affected. It is not necessarily symmetrical; it may affect one leg alone (*Barbadoes leg*), or the leg below the knee only, or the scrotum, ears, lips, arm, or the lower part of the abdominal wall. If the leg is affected, it becomes enlarged to two or three times its natural size. The skin looks œdematous, but it does not pit on pressure; it is obviously greatly thickened. Where folds naturally occur, as about the knee or ankle, there are deep sulci, and these may be moist from retained sweat or sebum. Pigment is increased in the limbs, the surface becomes rough and scaly, here and there are patches of hypertrophied papillæ, and, in other parts, vesicular or moniliform prominences due to dilated and varicose lymphatic vessels. Sometimes these burst, and discharge a more or less turbid lymph. The scrotum has sometimes grown to a tumour weighing more than one hundred pounds.

In tropical countries the disease often begins by attacks of lymphangeitis with swelling and redness like erysipelas, accompanied by fever (*elephantoid fever*), after the subsidence of which the leg is left bigger than normal. A fresh attack of fever and local inflammation after some months again leaves the leg worse than it was after the first, and the evil goes on increasing. Sometimes, however, the enlargement is gradual, and not at any time associated with febrile attacks.

In England erysipelas and phlegmasia alba dolens are occasionally causes of a permanent elephantoid change. Local pressure on veins and lymphatics may cause it, and I have seen it as a part of extreme obesity.

Anatomy.—The change affects chiefly the subcutaneous layer, which is enormously thickened by the growth of new connective tissue, partly gelatinous, but mostly in dense fibrous bands. The blood-vessels and lymphatics and lymphatic glands are much enlarged, and sometimes the nerves. The corium and epidermis are only slightly affected.

Treatment.—Very little can be done, except surgically; thus a large scrotum can be removed. But in any other part of the body such a measure is too serious. Bandaging, especially with rubber bandages, and elevation of the limb, may give temporary relief. Ligature of the artery has been tried and failed.

Other forms of filariasis.—The embryonal forms of *filaria diurna* are found in the blood in the daytime, and disappear at night; those of *filaria perstans* are in the blood day and night as well. The parental forms of these are not with certainty known. *Filaria medinensis* is well known as the *guinea worm*, which infests the subcutaneous tissues of the legs in certain parts of India and tropical Africa. This, the female worm, is from 12 to 30 inches in length and only one-tenth of an inch in width. It discharges its embryos into water, and they find an intermediate host in the small crustacean *Cyclops quadricornis*. It appears probable that it is transferred to man by means of drinking-water.

DISEASES OF THE BLOOD.

ANÆMIA.

The normal pink colour of the skin and deeper red colour of the mucous membranes are due to the blood circulating in their vessels: if a considerable loss of blood occurs—as, for instance, after injury, or from gastric ulcer, or during parturition—the natural colour is materially altered, and the skin becomes of a waxy whiteness, blanched, or bloodless, and even the lips and mucous membranes have only a very pale pink colour. This condition is spoken of as *anæmia*, and occurs under a great variety of circumstances, besides the direct loss of blood in quantity; moreover, it is accompanied in all its forms by numerous other disturbances, which directly result from the deficiencies which exist in the blood itself.

Causation.—A division, which after all may be artificial, is made between primary anæmia and secondary or symptomatic anæmia. In the latter, the bloodlessness results from direct losses of blood from the system, or from more or less definite diseases of particular organs, whereby the income or expenditure of the body is materially affected. In the former the change seems to depend on the blood itself, and cannot be traced back to any hæmorrhage, or to disease of any organ.

Secondary or *symptomatic anæmia* may be brought about in many ways. It includes (1) hæmorrhages of all kinds; these may be from incised or lacerated wounds, from epistaxis, gastric, intestinal (typhoid), or rectal ulceration, piles, excessive menstruation, uterine fibroid, ulcerating cancerous tumours, and other lesions that need not be mentioned. Many of these hæmorrhages are profuse, but do not recur, or only at long intervals; the anæmia is in direct proportion to the loss, and recovers perhaps quickly within a short time. Repeated small hæmorrhages may take place from piles, rectal ulcer, and uterine diseases, in which case the anæmia is persistent. Another form of hæmorrhage is that due to a diseased condition of the blood itself, such as occurs in purpura, scurvy, malarial poisoning, jaundice, and Bright's disease; but in some of these the anæmia is in part due to the condition of the blood which causes the hæmorrhage, and may be present before bleeding takes place. Anæmia is also caused by (2) continued discharges of pus from old sinuses, or leucorrhœal or other discharges in women; by (3) deficient ingestion of food, as in starvation or stricture of the œsophagus; and by (4) deficient assimilation, as in cancer of the stomach, of the liver, and of other assimilating organs. Assimilation is defective in the course of severe acute illnesses, such as enteric fever, pneumonia, pleurisy, and rheumatic fever; and pallor is a constant accompaniment of the later stages of the illness, and its early convalescence. Indeed, most chronic diseases affecting the viscera sufficiently interfere with blood-formation to cause anæmia—*e.g.*, phthisis, Bright's disease, aortic regurgitation, and all kinds of valvular disease in children. (5) Some other diseases affect the blood in a way that is not fully understood, and produce anæmia, such as lead-poisoning and syphilis. (6) A special group may be made of Hodgkin's disease, leuchæmia, and the intermediate forms in which the spleen, lymphatic glands, and marrow of the bones are diseased, since these organs are more or less concerned in the development of the red blood-corpuscles.

Primary or Essential Anæmia.—This may, perhaps, include cases of slight anæmia, which are either congenital or may be regarded as due to idiosyncracy, where the patient, if not so vigorous as others, is not materially affected in his health by the deficiency; but the two important varieties are (1) ***chlorosis***, or ***green sickness***, the anæmia which occurs so frequently in females about the time of puberty; and (2) ***pernicious*** or ***idiopathic anæmia***, which arises often without any obvious causes in adults, and is fatal in the majority of cases.

The red corpuscles are the essential elements of the blood, and their deficiency, which constitutes anæmia, may be brought about (1) by direct loss, as in various forms of hæmorrhage; (2) by a defect in their formation or development (*hæmogenesis*); and (3) by increased rapidity of destruction (*hæmolysis*). Many forms of symptomatic anæmia, as well as chlorosis, are due to defective hæmogenesis; pernicious anæmia appears to be the result of increased hæmolysis.

Symptoms of Anæmia.—Some features are common to all cases of pronounced anæmia, however caused. The skin is pale and waxy-looking. In cases of recent hæmorrhage the colour is almost white, but in pernicious anæmia there is a yellow tinge; in chlorosis a greenish tint is sometimes detectable, and a dirty yellow, earthy, or sallow tint in the anæmia of lead-poisoning, malaria, and syphilis. The lips are pale pink, and the cheeks may show a faint pink flush. The visible mucous membranes are pale pink, as seen in the mouth, the tongue, and the inner side of the eyelid. The altered colour of the blood is also manifest in the tint of the veins on the back of the hand, which show pink through the white skin, instead of dark purple through the pink skin. The patient is languid and weak, unfit for physical or mental exertion, and liable to headache, vertigo, the appearance of spots before the eyes, ringing in the ears, and attacks of syncope. In sudden and large losses of blood, such as occur in parturition, the anæmic condition of the brain may produce convulsions, but these do not occur in chronic cases, unless it may be near the fatal termination. There is dyspnœa on exertion, palpitation of the heart, and throbbing of the vessels; in some cases œdema of the feet may be present. The appetite is generally diminished, and the ingestion of food is often followed by weight or oppression at the epigastrium, or by severe cardialgia.

Any marked degree of anæmia is accompanied by murmurs over one or more of the cardiac orifices. The most common is a systolic murmur, often harsh in quality, heard loudest in the second left intercostal space, and traceable outwards along that space and towards the left clavicle—that is to say, in the area of the pulmonary artery. This murmur is often loudest in the recumbent position, and diminishes or even disappears when the patient stands up. In some cases a murmur is heard at the apex, and in more severe anæmia, systolic murmurs may be heard at all the orifices, or over the whole cardiac area, as well as behind. A similar murmur is often heard in the carotid vessels in the neck. Further, if the stethoscope be placed over the lower end of the jugular vein, at the point of separation of the sternal from the clavicular attachments of the sterno-mastoid, a continuous humming or rushing noise will be heard, which has been called the venous hum, or *bruit de diable*, from a French toy, called "diable," which makes a similar noise. This murmur is heard best in the erect posture, with the head turned away from the side which is being examined. Neither this nor the systolic pulmonary hæmic murmur is peculiar to anæmia, nor should either be regarded as evidence of anæmia; but they are almost constantly present in this condition, and are generally more marked in proportion to its intensity.

As to the manner in which the blood of anæmia causes these murmurs, there has been much discussion. The *bruit de diable* seems explicable on the assumption that the *quantity* of blood is less, by supposing that the upper part of the vein adapts itself to the lessened amount of blood passing through it, while the lowest part, or ampulla, is kept in its natural state of dilatation by cervical fascia. Blood then passes from a narrow space to a wider space beyond, fulfilling the requirements of the *veine fluide* theory (*see* p. 493). But it is not easy to show that the blood is less in quantity, and the very frequent occurrence of venous hum in children, quite independent of anæmia, does not seem clear on this hypothesis. The pulmonary hæmic murmur is still more difficult to explain. With the same theory in view, Fagge suggests that the base of the artery (and of the aorta) is unable to contract upon the diminished volume of blood to the same extent as the orifice through which the blood enters; and Goodhart states that in several conditions of anæmia the base of the pulmonary artery is morbidly dilated beyond the calibre of the orifice. By others the murmur is attributed to the action of the right ventricle. That this cavity is dilated is often shown by the visible pulsation in the second left intercostal space, corresponding to the conus arteriosus; and this advance of the ventricle is believed to force up and distort the pulmonary artery, producing an aneurysmal sacculation of its anterior wall. The increased loudness of the murmur in the recumbent position is compatible with this explanation.

The dilatation of the heart already mentioned carries the impulse into the line of the nipple, or even external to it. The pulse is commonly rather quicker than normal; generally soft, and rather full, but sometimes hard in slight cases; and the heart's action is readily quickened by exertion or excitement.

In severe degrees of anæmia hæmorrhages are liable to occur, and the temperature may be raised. Retinal hæmorrhages and pyrexia are, indeed, common in pernicious anæmia, but they are also seen in chlorosis and other forms occasionally.

Condition of the Blood.—The actual quantity of the blood in cases of anæmia is difficult to estimate; after any loss of blood, the vessels absorb liquid rapidly from the tissues, and thus are soon filled again with a blood the same or nearly the same in quantity as before, if deficient in corpuscles and chemical constituents. And in anæmia arising otherwise it is thought by many that the absolute quantity of the blood is not much affected. Quincke, however, has estimated, by the enumeration of blood-corpuscles before and after transfusion with normal blood, that the quantity in pernicious anæmia may be only 4 or 5 per cent. of the body-weight, instead of 7·7 per cent., as it is estimated to be in health.

To ascertain the *quality* of the blood, we have to examine it directly or by the microscope, to enumerate the corpuscles, and estimate the amount of hæmoglobin.

Enumeration of Corpuscles.—This is effected by the *hæmo-cytometer* of Thoma-Zeiss or that of Gowers, which differ only in detail. The former consists of a glass slide on which a "cell" is constructed one-tenth of a millimetre in depth and ruled at the bottom into squares, measuring $\frac{1}{20}$th mm. in the side, which are again ruled into groups of sixteen. In a specially-constructed pipette the blood is diluted to the extent of 1 to 100 by a saline solution (sodium phosphate or chloride) which does not injure the corpuscles; and a drop of the dilution is placed in the cell and covered with thin glass. The corpuscles settle down upon the ruled squares, each of which corresponds to $\frac{1}{4000}$ cubic mm. The corpuscles in several groups of 16 squares are counted, and the total, multiplied by 100 (the dilution) and 4000 (the size of the cell), and divided by the number of squares counted, gives the corpuscles in a cubic millimetre. In Gowers' instrument the squares are larger and the dilution is greater, but the principle is the same. If the diluting fluid also stains the leucocytes (*e.g.*, by methyl-violet) these can be better counted at the same time.

Estimation of Hæmoglobin.—For this Gowers' *hæmoglobinometer* and Fleischl's *hæmometer* are available. In the former, two tubes are provided, one of which contains a pale pink substance to act as a standard. The other is graduated to 100 degrees, and in this a measured quantity of blood is diluted till it matches the colour of the standard. The figure on the scale which the solution then reaches represents the percentage amount of hæmoglobin. In Fleischl's apparatus the diluted blood in a glass-bottomed cell is compared with the increasing thickness of a wedge of coloured glass used as a standard.

The percentage of hæmoglobin may be less than that of the corpuscles, if individual corpuscles are smaller, or contain less pigment than normal: the hæmoglobin percentage is greater if the corpuscles are larger, or contain more pigment than normal.

Microscopic Examination.—This can only be done satisfactorily after the corpuscles have been stained. The blood is spread on a glass slide, or cover-glass, in a thin layer, or film, dried, "fixed" by heat or chemical means, and stained by various dyes, which may be acid (eosin), alkaline (fuchsin, methylene-blue), or neutral (a mixture of the two others).

The different corpuscles which may be seen in health and disease may be now enumerated. Red Corpuscles, or Erythrocytes:—The normal red cell measuring in diameter 7·5 μ; small corpuscles, or *microcytes*, from 2–6 μ; large corpuscles, or *megalocytes*, from 8–15 μ; misshapen, distorted, often pear-shaped corpuscles, or *poikilocytes;* nucleated red corpuscles (*erythroblasts*), divided according to their size into *normoblasts*, *microblasts*, and *megaloblasts*, or *gigantoblasts*.

The leucocytes present even in health many varieties; they are distinguished by their size, the number of their nuclei, and by the reaction to dyes of the granules in the cell-body. In relation to this point, the granules of some cells stain with acid dyes (*oxyphile* or *eosinophile* cells), of others with basic or neutral stains (*basophile*, *neutrophile*). In normal blood the majority (60 to 70 per cent.) of the leucocytes are *multinuclear neutrophile*, measuring about 13·5 μ; there is a much smaller number (15 to 25 per cent.) of *small uninuclear cells*, measuring 5–10 μ, and a very small number of *large uninuclear* cells, 13–15 μ in size, and *oxyphile* or *eosinophile* cells, containing large granules staining deeply with eosin.

Marrow-cells or myelocytes occur normally in the red marrow of bones; but they are found in the blood only in diseased conditions. The myelocyte is of large size, 20 μ in diameter, with neutrophile granules, and a large nucleus, staining very feebly.

Blood-plates are round or oval bodies, found in normal blood. They are 2 to 3·5 μ in diameter, faint yellow and granular in appearance, adhesive, and thus readily clinging to blood-corpuscles and to one another.

Degeneration of the red blood corpuscles is shown by the tint of the hæmoglobin becoming fainter from the centre outwards, or by the more irregular appearances of vacuolation, and by irregular points and knobs forming on the corpuscle, which gets deformed and so forms a poikilocyte. Attempts at the *regeneration* of the blood are believed to be indicated by the presence of nucleated red blood cells, especially normoblasts.

The **Anatomical Changes** found after death by anæmia are best illustrated by cases of pernicious anæmia (*see* p. 752). The organs are universally bloodless, and in some cases of secondary anæmia is found the fatty degeneration of the muscular fibres of the heart (tabby-cat striation) which is constant in the pernicious forms. It is no doubt due to an imperfect process of oxygenation, which is the result of a less quantity of hæmoglobin.

CHLOROSIS.

This name—or its English equivalent, green sickness—is applied to a form of anæmia which especially occurs in girls and young women between the ages of fourteen and twenty-four, though a similar condition is exceptionally seen in boys. In its milder varieties, it presents the characteristics already described; in its severer forms, it may be difficult to distinguish from pernicious anæmia. Languor, feebleness, dyspnœa, and palpitation on exertion, vertigo, headache, noises in the ears, spots before the eyes, nausea, eructations, pain after food, and constipation, occur just as in cases of secondary anæmia. The patient may present all degrees of pallor, often with some temporary flushing of the cheeks or lips; sometimes also, no doubt, there is a tint which justifies the name chlorosis, but it can scarcely be recognised in all cases—Zimmermann says it occurs only in those of a dark complexion. Both blood-corpuscles and hæmoglobin are diminished, but the former are rarely below 60 per cent. of the normal, whereas the hæmoglobin may be only 25 or 30 per cent. of the normal!? Thus each corpuscle has on an average less than its normal amount of hæmoglobin. This relation of corpuscles to hæmoglobin is often called the *chlorotic type;* but it occurs also in other forms of anæmia. Microcytes and poikilocytes may be found in varying quantities. There are murmurs at the base of the heart and over the jugular veins. The pulse is not necessarily feeble; its tension may be, but is not always, increased. An almost constant symptom, which more than all attracts the attention of the patient or her friends, is the suppression or diminution of the menstrual flow. In the younger patients the menses may never have appeared, and may continue absent as long as the chlorosis lasts; in those who have already menstruated, the flow becomes scanty, pale, or irregular, or ceases altogether. Only occasionally are the menses more abundant than normal. It is possibly in connection with these menstrual disorders that the mental condition of the patient is often markedly affected, as shown in irritability and a tendency to hysterical manifestations. Constipation is frequent, and the urine is pale. Exceptionally optic neuritis occurs and may be followed by atrophy; and thrombosis is liable to occur and may be fatal if it affects the cerebral sinuses.

Pathology.—Numerous hypotheses have been put forward to explain the occurrence of chlorosis. The following are some of them:—(1) Disorders of menstruation, especially amenorrhœa; (2) Congenital narrowness of the aorta, which was found pathologically by Virchow, and is referred now to a hypoplasia of the mesoblastic layer; (3) Disorders of the stomach, amongst which are (*a*) excessive acidity of the gastric secretions, (*b*) proptosis of the stomach, (*c*) gastric ulcer and hæmorrhage. Tight lacing has been considered a possible cause of proptosis and hæmorrhage; (4) The presence in the blood of unassimilable forms of iron, from deficiency of hydrochloric acid in the system (Zander); (5) Disorders of the intestine, especially habitual constipation with retention of fæces followed by absorption of ptomaines or other toxic substances. Bunge thought that the organic iron compounds of the food (*hæmatogen*) were broken up by decomposition in retained fæces and assumed forms less capable of being absorbed; (6) Lloyd Jones states that naturally at puberty the specific gravity of the blood becomes greater in males and less in females; and that this fall in females is due to a diminution of the hæmoglobin. He thinks that chlorosis is an aggravation of this natural process. Undoubtedly there must be some connection between the sex, puberty, and the onset of chlorosis; but amenorrhœa is a consequence and not a cause. The essential change in the blood, that is, the want of hæmoglobin, shows that the fault lies in a defective hæmogenesis; and so far no completely satisfactory explanation has been afforded of this defect.

Diagnosis. —The recognition of chlorosis is generally easy; but it may be difficult, in some severe cases where the heart is dilated and murmurs of mitral regurgitation are heard, to decide whether the cardiac lesion is primary, or a result of the anæmia. Amenorrhœa, the absence of rheumatism, scarlatina, or chorea in the history, and extensive præcordial murmurs, rather than one localised to the heart's impulse and the back, would be in favour of chlorosis. The presence of any primary lesion (*see* p. 745) must always be carefully excluded.

Prognosis.—Chlorosis is rarely fatal; it often lasts months or years, and frequently recurs after cure.

Treatment.—For the restoration of the blood-state in chlorosis, the use of iron is of the first importance. This may be given in several forms: if there is irritability of the stomach, the less astringent forms are advisable, such as reduced iron, 2 or 3 grains three times a day, the ammonio-citrate, or the tartrate, 5 to 10 grains. The more astringent forms, however, when they can be borne, are more quickly efficient—*e.g.*, the perchloride, 10 to 20 minims of the tincture, or the sulphate, from 3 to 5 grains. A well-known remedy is Blaud's pill, containing 2½ grains of sulphate of iron, and the same of carbonate of potassium. One, two, or three pills are given thrice daily. The Pilula Ferri, B.P., is a similar combination. I have found that the much-lauded Griffiths' mixture often makes patients sick. Iron should be always given immediately after meals. According to Bunge, iron salts are not absorbed directly from the intestinal canal, but they prevent decomposition of the above-mentioned *hæmatogen*. It is always desirable to combine some laxatives with the iron, both to counteract its astringency, as well as to overcome the natural tendency to constipation. This may be done by the use of sulphate of magnesia, in combination with the perchloride or the sulphate; or by the use of aloes, in the form of aloes and iron pill; or an aloes or myrrh pill may be given at night with the iron mixture. In some cases arsenic may be of value in combination with iron. The diet must be good and nourishing, and may be modified somewhat according to the fat or lean condition of the patient. In severe or advanced cases, treatment by drugs may fail entirely until the patient has prolonged rest on the couch, or even in bed. Exercise increases both the tendency to dilatation of the heart and the overstrain upon the blood-forming organs; and, though fresh air is desirable, it must be obtained without exertion on the part of the patient.

Idiopathic or Pernicious Anæmia.

Ætiology.—This curious disease, first spoken of as *idiopathic anæmia* by Addison, and others after him, later as *progressive pernicious anæmia* by Continental writers, affects both sexes equally, and is most frequently seen between the ages of twenty-five and forty. Its origin is often quite obscure; pregnancy and the puerperal state have been credited with its causation in many cases, and gastro-intestinal disturbance, privation and mental shock in others. Some fatal cases of anæmia following at long intervals upon a large hæmorrhage have also been recorded, but it is doubtful whether these should be included in the present category.

Symptoms.—The patient with pernicious anæmia gradually loses strength and becomes paler; his skin acquires a yellow tint, rather than the waxy white of ordinary anæmia. At the same time, even with extreme anæmia, the patient does not lose flesh, and the subcutaneous fat may be abundant. There are the same languor, indisposition for mental and physical exertion, giddiness, noises in the ears, &c., which have been described in the other forms, as well as dyspnœa on exertion, cardiac palpitation, and vascular murmurs, The patient complains of dryness of the mouth and throat, loss of appetite and nausea; and in a large proportion of cases there are vomiting and diarrhœa. The urine is usually high-coloured from excess of urobilin, and free from albumen. It may contain a full quantity of urea and uric acid, as well as indican. The bones are often tender on percussion or pressure. The pupils are mostly dilated; and the retinæ show numerous small hæmorrhages, which are abundant round the optic disc. These are striated, or flame-shaped, and may be accompanied with white spots. Another feature of idiopathic anæmia is the presence of fever, which may give a temperature of 101° or 102°, but is generally irregular. It may be absent for days together, and the temperature is often subnormal before death. The blood is excessively pale, and the red corpuscles are reduced to 2,500,000 per cubic mm., or even to 500,000 or lower; but the diminution of the hæmoglobin is less in proportion. This contrasts with the condition in chlorosis, and is due to the fact that individual corpuscles are relatively large and contain excess of pigment. Megalocytes are common, and poikilocytes are so numerous that their presence was regarded at one time as the distinguishing feature of the disease. Usually there are some nucleated red cells, especially megaloblasts, but the leucocytes are not materially affected.

Anatomical Changes.—Besides the universal pallor of the organs, one of the most constant conditions *post mortem* is fatty degeneration of the heart, principally in the form of the striation already mentioned. It occurs in the left ventricle and on the papillary muscles. There is also fatty degeneration of the liver and kidneys, and of the intima of the arteries. Hæmorrhages are found not only in the retina, where they have been seen during life, but in the serous membranes, the endocardium, the mucous membrane of the stomach, the lungs, the surface of the brain, and other parts. Fenwick and others have found fatty degeneration or atrophy of the tubular glands of the stomach, and infiltration of leucocytes between the tubules. The spleen is sometimes slightly enlarged. The marrow of the bones has been found of a reddish purple colour, with large numbers of nucleated red corpuscles, especially megaloblasts, while the fat cells are all or nearly all destroyed—a return to the condition of the embryo. There is, moreover, an abundant deposit of iron in the cells of the liver and in the spleen, which in the liver is most abundant in the peripheral zones of the lobules.

The **Pathology** of idiopathic anæmia is still not well understood. Probably the changes in the marrow of the bones, to which some have referred the disease, are secondary; and the atrophied condition of the gastric glands seems scarcely sufficient to account for it. The excess of urobilin in the urine, and the deposit of iron in the tissues, suggest that the immediate cause of the anæmia is the destruction of red corpuscles in the blood (*hæmolysis*), though how this is brought about remains open to question. W. Hunter believes that this destruction takes place within the portal circulation, and that it is due to the poisonous action of cadaveric alkaloids. Micro-organisms have been discovered in the blood by Klebs and Frankenhäuser; but the observations want confirmation.

Exceptionally, patients with pernicious anæmia have suffered from nervous symptoms, suggestive of spinal sclerosis, such as numbness, weakness, ataxy, loss of knee-jerks, &c.; and sclerosis of the posterior and lateral columns has been found after death.

Diagnosis.—In any supposed case of *idiopathic anæmia* it is important to search most carefully for organic disease, such as cancer, which might be the cause of the bloodlessness. The characteristics upon which the diagnosis depends are the absence of such cause, the retinal hæmorrhages, the absence of wasting, and the condition of the blood as to corpuscles and hæmoglobin. The latter with the dark colour of the urine will serve to distinguish it from chlorosis.

Prognosis.—This is very unfavourable, but Pye-Smith has collected twenty cases of recovery, as compared with 103 fatal cases. Of the fatal cases a large number die in about six months, whereas others last twelve or fifteen months.

Treatment.—Arsenic seems to be the most efficient remedy of all drugs that have been tried; and it should be pushed to full doses. Fresh bone-marrow of the ox or calf (3 ounces daily), and antiseptics, such as salol, β-naphthol, and salicylate of bismuth appear to have been successful in a few cases. Some have recovered after transfusion, but in others this has proved of no avail. Iron has generally no effect, and other tonics are equally useless. The patient should be placed under suitable hygienic conditions, the diet should not be too highly nitrogenous, and special symptoms should be treated as they arise.

SECONDARY ANÆMIA.

The condition of the blood in secondary anæmia is somewhat like that of chlorosis. The corpuscles may not be very much diminished in number, but the loss of hæmoglobin is relatively greater. Microcytes as well as poikilocytes occur in the severe cases, and in the worst cases nucleated red cells, especially normoblasts. The leucocytes are variable; they are often increased in number, especially in cases dependent on inflammatory and pyogenic conditions.

LEUCHÆMIA.

(*Leucocythæmia.*)

In this disease there is a great increase in the number of white corpuscles in the blood, associated with changes in the spleen, the lymphatic glands, or the marrow of the bones. As is well known, the proportion of white blood-cells, or leucocytes, in the blood varies considerably in health, and it may rise to an unusual degree in some morbid conditions, such as irritation of the lymph-glands, in fevers, and in pregnancy. This may be called ***leucocytosis***; but it is temporary, relatively slight in amount, and the increase chiefly concerns the multinuclear neutrophiles common in normal blood.

In well-marked examples of leuchæmia the blood is pale and thin as it issues from a wound, and as seen after death it is grumous-looking, or forms pale pus-like clots; its coagulation, moreover, is imperfect from the fibrin being deposited in a granular, rather than in a fibrous, form. Under the microscope the white corpuscles are seen to occupy nearly the whole of the field, instead of being few and scattered as in health; on the other hand, the red corpuscles are less numerous than in health. If the hæmacytometer be used, the white corpuscles are found to number from 200,000 to 700,000 in the cubic mm., instead of 7000 or 8000; and the red corpuscles may be from 2,000,000 to

3,500,000—*i.e.*, only 40 to 70 per cent. of the normal. The proportion of the white to the red corpuscles is in different cases between 1 : 10 and 1 : 1. The hæmoglobin is somewhat less in proportion than the red corpuscles, as is seen in chlorosis.

Varieties of Leuchæmia.—The division of leuchæmia into two chief forms is justified partly by the difference in the organs diseased at the same time, but especially by the different conditions of the blood, in relation to its leucocytes, which are found in these circumstances. In *spleno-medullary leuchæmia* the spleen is enormously enlarged, the bone-marrow is diseased, and the lymphatic glands are little if at all affected. The leucocytes are very greatly increased, and reach the highest amount found in leuchæmia. From a third to a half of these leucocytes are *myelocytes* or *marrow-cells*, which do not occur in the normal blood. *Eosinophile cells* are also very numerous, and many of these are myelocytes. The multinuclear or polymorphonuclear leucocytes are increased in number, though less relatively to the others. The red corpuscles show variations in shape and size; and in addition the nucleated red cells, mostly *normoblasts*, are numerous. Charcot-Leyden crystals (*see* p. 428) are present. In *lymphatic leuchæmia*, which is a much less common form of the disease, the lymph-glands all over the body are enlarged in size; but the bone-marrow may be also affected, and the spleen may show a slight enlargement. In the blood the leucocytes are less numerous than in the spleno-medullary form, but they consist almost entirely of the small uninucleated leucocyte, with a large nucleus, and scarcely any protoplasm visible around it. Erythroblasts and eosinophile cells are absent, and myelocytes are only occasionally present.

The **Pathology** of leuchæmia is still debateable. The opposed views are: (1) that the increase of leucocytes is due to excessive proliferation in the blood-forming organs, the spleen or marrow and lymph-glands respectively; (2) that the increase of myelocytes in one case, and of lymphocytes in the other, is primary, and the enlargement of the various organs is the result of the accumulation of the leucocytes within them.

SPLENO-MEDULLARY LEUCHÆMIA.

Ætiology.—The cause of spleno-medullary leuchæmia is often difficult to trace. The disease occurs in men more often than in women, and mostly in middle life, though sometimes in quite young children. Deficient hygienic conditions, the disturbances of pregnancy, the climacteric in women, and occasionally injuries to the spleen, have been thought to have some influence; but intermittent fever holds the first place as a remote cause of leuchæmia. Gowers found a history of ague in thirty out of 150 cases of which he analysed the records, and others have noticed the same fact;

but there is often a long interval between the two diseases, and the way in which the one produces the other is quite unknown.

Symptoms.—One of the first indications of leuchæmia, in a great number of cases, is the swelling of the abdomen from the *enlargement of the spleen*, which may have been developing for some time without giving any sign. It may then be found occupying the whole of the left side of the abdomen, forming a firm, hard tumour, which extends backwards into the flank, while its anterior margin begins about the ninth costal cartilage, reaches the middle line at the umbilicus level, and not infrequently below this extends two or three inches to the right. This anterior margin is more or less sharp, and presents one or two notches. Fagge first pointed out that the position taken by the spleen in these cases was determined by its attachment to its vessels, which compelled it, so to say, to enlarge along the circumference of a circle of which the cœliac axis is a centre. In earlier stages the spleen simply occupies the left hypochondriac region, similar to the enlargements in ague, and in some cases of typhoid fever. The *liver* is moderately enlarged, and can be felt for one or two inches below the right costal margin. The implication of the *bone-marrow* may be shown by tenderness on pressure or percussion of the corresponding bone.

The general effects of the illness do not always show themselves early; especially it must be observed that when the spleen is already very large, and the leuchæmia unmistakable, there may be no pallor of the skin, lips, and mucous membranes. Later on, however, the patient loses colour, and becomes sallow, or markedly anæmic.

The *temperature* is generally affected in this disease; there is either continuous moderate pyrexia, or there are periods of pyrexia alternating with periods of apyrexia; and the febrile reaction sometimes gives a flush to the skin, which may help to mask the approaching anæmia.

The altered condition of the blood shows itself in the occurrence of *dyspnœa* and of *hæmorrhages*, which last chiefly take the form of epistaxis, bleeding from the gums and mouth, and purpuric spots under the skin; but also occasionally bleeding from the lungs, stomach and intestines, kidneys or uterus, or hæmorrhage into the brain. Hæmorrhages also occur in the retina, where they may be seen with the ophthalmoscope associated with white streaks and spots, said to consist of masses of leucocytes; and the retinal veins are often remarkably tortuous. Other organs may be affected, but the changes are chiefly observed after death. The urine is acid, of high specific gravity, and the urea and uric acid are in excess. Indican is often present. Albumen is rare unless the kidneys are diseased.

The course of the disease is generally progressive until its termination in death, and it lasts from two to four years. Towards the end the pallor increases, the feet and other parts of the body become œdematous, ascites and hydrothorax may be added, the pulse is quickened, and palpitation is frequent. Diarrhœa is occasionally a prominent symptom. Finally, death takes place from loss of blood, asthenia, diarrhœa, pleurisy, pneumonia, bronchitis, or cardiac failure from dilatation; and occasionally from cerebral hæmorrhage.

Anatomical Changes.—The *spleen*, in cases of splenic leuchæmia, varies in weight from one to eighteen pounds, the average of seventy-two cases analysed by Gowers being six pounds. It is uniformly enlarged, and presents its normal shape; on the surface are often patches of thickening of the capsule, and the organ is more or less adherent to the abdominal wall, diaphragm, or adjacent viscera. On section it often has a brownish, rather than a red colour, homogeneous, or marked with paler lines due to thickened trabeculæ. It is smooth, hard, and dry. Not infrequently there are large wedge-shaped infarcts, yellow in colour if old, red if recent. The change in the spleen itself is one of great increase of the splenic pulp, which is full of the same cells as are found in the blood; and in long-standing cases the stroma becomes markedly fibrous, and the trabeculæ are increased. Charcot-Leyden crystals are often found abundantly in the spleen.

The *liver* is enlarged, and may reach twice or three times its normal size. It is pale and smooth, and presents under the microscope a dense infiltration with leucocytes, which for the most part surround the portal vessels in their distribution, but are partly in a nodular form. The vessels also are full of leucocytes. The *kidneys* are pale, and enlarged from granular degeneration of the cells, and distension from leucocytes; or they present greyish-white deposits, running like striæ between the cortical tubules. There may be stomatitis, or pharyngitis, swelling of the *tonsils*, and of the *follicles* at the root of the tongue; and swelling and superficial ulceration of the follicles of the intestine. The *thymus*, *thyroid*, and *supra-renal bodies* have also been found diseased, and tumours in the skin have been recorded. Sometimes the *lungs* present hæmorrhagic infarcts. The *marrow* of the bones is either yellow and pus-like, or pink and firmer, the fat of the marrow being replaced by a tissue like that of active marrow, in which marrow-cells and nucleated red cells are abundant.

Diagnosis.—The spleen is easily recognised by its position on the left side, extending from the ribs to the pelvis, with its irregular anterior margin; but the blood must be examined for excess of leucocytes in order to distinguish the disease from *splenic anæmia* (p. 729). In women the cases have not infrequently been mistaken for *ovarian tumours;* but it should be remembered that an ovarian tumour large enough to reach the ribs is central in position, while the history will show that it began in the lower part of the abdomen. On the other hand, the enlarged spleen may sometimes be felt in the pelvis behind the uterus. In earlier stages, where the spleen is small, and the number of white blood-corpuscles is not yet in great excess, it may not be easy to come to a conclusion; but in the absence of other lesions, a provisional diagnosis of splenic leuchæmia must be made, and the case treated accordingly. No absence of pallor should deter one from examining the blood, as the patient may have a good colour when the spleen is enormous, and the leuchæmia pronounced.

The **Prognosis** is unfavourable, and recovery is very rare.

Treatment.—Arsenic is the remedy that seems to have given most promise; it must be used perseveringly, and in increasing doses as long as it can be borne. Mosler has injected arsenic into the substance of the spleen. He has also obtained good results from quinine given by the mouth, though others speak of it as useless. Iron is generally regarded as of no value, and the influence of cod-liver oil is doubtful. Inhalations of oxygen (one to two cubic feet daily) are believed to have done good. The local application to the spleen of the ice-bag, cold douche, or electricity (galvanic current) may reduce its size. Excision of the spleen has been tried, with a quickly fatal result, from collapse or hæmorrhage. The disease cannot be arrested in its later stages. Gowers urges as a preventive that in cases of enlarged spleen remaining after ague, every effort should be made to restore the organ to its normal condition, by the avoidance of cold, injury, and all causes of portal congestion, and the use of quinine, ergotin, local affusion, and galvanism.

LYMPHATIC LEUCHÆMIA.

This is of much rarer occurrence than the spleno-medullary form. Its ætiology is still more obscure, its general symptoms very similar, but its course is usually more rapid. The lymphatic glands all over the body are affected, and they may be felt in the neck, groins, or axilla. They are moderately large, not very hard, and move freely upon one another. The mesenteric glands are even more often enlarged than the above; the retro-peritoneal, thoracic, portal, and iliac glands less so. The spleen may be a little enlarged, and the bone-marrow and thymus are sometimes involved. On section the glands are whitish-pink in colour, and microscopically are found to be distended with the uninucleated leucocytes found in the blood. The bone-marrow is full of the same leucocytes. Some very rapid cases of *acute leuchæmia* have been recorded (Fraenkel, Bradford) which are probably allied to lymphatic form. The lymph-glands are slightly enlarged, the leucocytes in the blood are chiefly lymphocytes, the bone-marrow is diseased, containing numerous lymphocytes, and the patients have purpuric hæmorrhages, and severe stomatitis, so that a confusion with purpura and scurvy may readily occur.

PURPURA.

This term is applied to a diseased condition in which a number of hæmorrhages occur under the skin, so as to produce blotches of a more or less purple colour. It has been already seen that there are similar hæmorrhages in a number of diseases, either from an altered state of the blood, or from mechanical interference with its circulation ; for instance, in scarlatina, measles, variola, typhus, cerebro-spinal fever, and the plague; in leuchæmia, Hodgkin's disease, cirrhosis of the liver, and Bright's disease; and in malignant endocarditis, and other diseases of the heart. A purpura also occurs in scurvy or hæmophilia, in some cases of overdosing with potassium iodide (*iodic purpura*), and in some nervous diseases, *e.g.*, tabes dorsalis. In all these cases it is clearly recognised that the hæmorrhage is secondary, and it is often called *symptomatic.* But in other cases the disease occurs spontaneously and without obvious antecedent in persons practically healthy.

The most familiar forms are *purpura simplex* and *purpura hæmorrhagica*, which appear to be essentially the same thing in different degrees of severity.

Symptoms.—In its mildest forms (*P. simplex*) purpura consists simply in the appearance of spots of a dull red, deep red, or bluish-purple colour in different parts of the body. They are circular, vary in diameter from a millimetre to a third of an inch, do not disappear on pressure, and are generally, when of this small size, not raised above the surface. They have no special relation to the position of the hairs. In some cases they occur only on the feet and legs, but in others are scattered uniformly, or, at any rate, indiscriminately. Each spot fades after a time, becoming brownish or yellow in tint, and the larger patches go obviously through the changes characteristic of a bruise. Very little constitutional disturbance accompanies the eruption: the patient may be pale, and loses appetite. Recovery generally takes place in from ten to twenty days.

In severe cases the hæmorrhages are more extensive, the skin may be raised by large masses of blood beneath it, and bleeding takes place from the various mucous membranes (*P. hæmorrhagica*). The nose, mouth, stomach and intestines, the kidneys, the female genital organs, and occasionally the bronchial mucous membrane may thus be the source of the blood. The gums are never swollen as in scurvy, but sometimes a spot of hæmorrhage is seen in their substance. If the loss of blood is considerable the patient becomes anæmic, and in the severer stage there may be some rise of temperature, and a stage of prostration ensues which terminates in death. Indeed, the hæmorrhagic forms are very often fatal, and *post-mortem* examination may reveal other ecchymoses in nearly all the mucous membranes, in the pelvis of the kidney, in the pleura, pericardium, peritoneum, in the meninges, and even in the lungs, and the medulla of the bones. A cerebral hæmorrhage may be the cause of death. Sloughing and ulceration of the mucous membrane of the bowels have also been found, leading to perforation and peritonitis.

Purpura rheumatica (*Peliosis rheumatica* or *Schönlein's disease*) is regarded by some as a hæmorrhagic erythema occurring during acute rheumatism, or in a rheumatic patient; while by others the arthritis is held to be the result of the purpura or its antecedent. Osler includes under the term any case in which purpura, erythema exudativum, purpura urticans, or urticaria, is associated with multiple arthritis. The illness may set in with sore throat, and pyrexia, occasionally causes endocarditis or pericarditis, and is very liable to relapse. S. Mackenzie limits the term to actual hæmorrhages, which may occur during rheumatism or in rheumatic subjects; beginning in the lower extremities, worse in the evening or reappearing then after improvement, persisting for long periods, but rarely fatal.

In *Henoch's purpura*, the lesion of the skin, which may be erythematous or urticarious as well as hæmorrhagic, is accompanied by attacks of abdominal pain, vomiting and hæmorrhage from the bowel. It occurs in children and recurs frequently during weeks or months.

Pathology.—Little is really known of the pathology of purpura. The proximate cause must be in the blood or the minute vessels; the former presents chiefly the characters usually found in secondary anæmia. Suggestive changes have been found in the vessels in some cases, and bacteria have been found in the tissues or vessels, but their relation to the disease is not clear.

Diagnosis.—In making the diagnosis, all the possible causes of a petechial eruption above mentioned must be excluded. *Scurvy* is distinguished by the spongy condition of the gums, the subcutaneous or fascial indurations, the greater degree of ill-health, and generally by its causation. *Malignant sarcomatous* growths may present some resemblance to purpura hæmorrhagica. It is well also to remember that the children of the poor sometimes present extensive petechial eruptions as the result of *fleabites*. The spots are uniformly about the size of a pin's head, and all disappear entirely after a few days in better circumstances.

Treatment.—In milder cases, rest in bed, tonic medicines, and good simple food will often rapidly effect a cure. Iron, arsenic, and quinine may be given in the usual doses. Where the purpura affects the lower extremities chiefly it often disappears directly the patient takes to bed, and returns if walking about is too hastily resumed. In severe cases, also, arsenic may be given; but if hæmorrhage take place from the mucous membranes, astringents must be employed, such as turpentine, acetate of lead, ergotin, gallic acid, or dilute sulphuric acid. Turpentine (10 minims three times a day) is especially recommended for *P. rheumatica* by S. Mackenzie. Calcium chloride (20 grains every 3 or 4 hours, subsequently reduced to 15 or 10 grains) is stated to increase the coagulability of the blood, and may be tried.

SCORBUTUS.

(*Scurvy.*)

Scorbutus is characterised by a profound change in the blood, resulting in hæmorrhages under the skin and in other parts of the body, a spongy condition of the gums, anæmia, and prostration.

Ætiology.—It has been abundantly proved that the essential cause of this disease is the deprivation of fresh vegetable food. It may occur in either sex and at any age, and is clearly not contagious in its origin; but it has arisen over and over again under circumstances entailing a restriction of the dietary in respect of vegetables. Thus, it has been in past times the scourge of sailors on long voyages, so that it is frequently spoken of as sea-scurvy, though such a term does not now distinguish it from any other form; and it has severely affected armies and other large collections of individuals, such as those in prisons, and sometimes even in hospitals. It is still more frequently seen amongst sailors; but cases occasionally happen amongst those who can get vegetables, if for any reason, such as poverty, or dyspeptic troubles, they have habitually abstained from eating them.

The influence of this wrongful diet in producing the disease is aggravated by several other depressing circumstances which not infrequently co-exist, such as damp, cold, fatigue, drink, want of sunlight, as well as prostration from wounds, ague, dysentery, or syphilis. Naturally, the particular constituents of the vegetables, which are necessary to prevent its occurrence, have been the subject of investigation. Sir A. Garrod has maintained the theory that it is the potassium salts which are requisite; the value of lemon-juice in the treatment of scurvy suggests that citric acid is wanted. Neither view seems of itself to be correct, but possibly the defective elements are the salts of potassium with the organic acids, citric, tartaric, and malic. Ralfe suggested that the disease is primarily a diminished alkalinity of the blood; the urine contains more uric acid, but less alkaline phosphates, and is less acid.

Symptoms.—The disease generally comes on insidiously. The patient loses colour, becomes weak, languid, drowsy, or apathetic, and complains of flying pains in the loins or limbs. After a time—it may be a week or more—petechiæ appear upon the skin of the lower extremities and other parts of the body, and as a rule each hæmorrhage is situated around the base of a hair. The spots are small, red or reddish-brown, and not raised above the surface. Some others appear which more or less resemble bruises produced by violence, and large wheals or *vibices* may also be present. These various hæmorrhages occur all over the body; and there may be large extravasations of blood in the eyelids, or even subconjunctival ecchymosis, though very often the face is spared. Associated with this purpuric condition must be mentioned the occurrence of tense, brawny indurated swellings in different parts of the body, especially in the popliteal space, the bend of the elbow, under the angle of the jaw, and in front of the tibia: these are due to effusions of blood, or blood-stained fibrin, or simply pale yellow fibrinous material, under the fascia, or between the muscular bundles, or between the periosteum and the bone.

Another feature which is commonly regarded as constant, is the condition of the gums. These become swollen, fleshy, or spongy, detached from the teeth, and projecting beyond them in loose, bluish-red masses, which are painful, and bleed on the slightest touch. The teeth become loosened, the patient is unable to chew, and the breath is fœtid. The swelling of the gums may be so great that they project from the lips, and ulceration often results. The rest of the mouth is not affected in the same way. The tongue is large and indented. Sometimes—and this is insisted on by Buzzard—the gums do not become spongy, but are only pale; and in all cases the change seems to be determined by the presence of teeth, so that it is absent where there is a gap in the series, and in toothless infants and old people.

When all these changes have developed, the patient has a sallow, bloated look, is markedly breathless on exertion, though no physical signs may be detected in the lungs; is subject to fits of syncope; and is totally unfit for bodily or mental exertion. The temperature, however, is generally not raised; the pulse is variable; and the urine is usually free from albumen. Hæmorrhage from the mucous surfaces, especially epistaxis, is not uncommon; and the feet are often œdematous.

In more serious cases there is hæmorrhage from the stomach and intestines, or from the lungs; pneumonia, gangrene of the lung, pericarditis, or pleurisy, which may be hæmorrhagic; or enlargement of the spleen and albuminuria. The skin over blood-extravasations may slough from pressure or irritation, and leave fungoid and very offensive ulcers. Dysentery sometimes complicates scurvy, but is generally regarded as having an independent origin. A curious condition of vision known as *hemeralopia* frequently occurs, and may be one of the early symptoms. The patient can see clearly and well in the daytime, but in the dusk, or the darkness of night, becomes quite blind, and cannot see his way about. The ophthalmoscope shows no change in the eye, and normal sight is restored as the scurvy is cured.

Death takes place from increasing exhaustion, with anæmia and emaciation generally, after many weeks. But it may occur more quickly from sudden syncope, from pneumonia or gangrene of the lung, from hæmorrhagic inflammation of the serous membranes, or from cerebral hæmorrhage. In cases that recover, the improvement under suitable treatment is at once manifest, and often very rapid; but it is stated that the deeper effusions may leave thickening and fibrous bands, as a result of which the limbs are partly contracted, and the corresponding muscles are atrophied. Sometimes the joints are ankylosed.

Occasionally scurvy is seen in children under two years old (*infantile scurvy*). It is traceable to the absence of fresh milk from the food, and is often associated with rickets. It is characterised by earthy pallor, emaciation, spongy gums, œdema of the feet, tenderness and swelling of the legs referable to hæmorrhage under the periosteum, and separation of the ends of the bones, indicated by crepitus.

Anatomical Changes.—In fatal cases the lesions are found which have been mostly manifest during life—*e.g.*, the blood extravasations in the skin, and the effusions, whether sanguineous or fibrinous, in the aponeurotic sheaths, and under the periosteum; and in infants separation of the epiphyses. Occasionally hæmorrhage has occurred on the surface or in the substance of the brain. Frequently the pleural cavities contain serum, and there may be engorgement of the lung with serum or blood; sometimes it is even gangrenous. Hæmorrhages may also take place into the cardiac muscle, into the pericardium, or into the mucous membrane of the stomach and intestine: these latter may cause abrasion or ulceration. The liver and spleen are often large, filled with blood, soft, and friable; and an acute nephritis is described as occurring in severe cases. The blood presents no specific character; it is like that of secondary anæmia.

Diagnosis.—There can be little difficulty in recognising this disease when the circumstances are such as would be likely to lead to it: but the diagnosis requires care in isolated cases. It is distinguished from *purpura* by the general illness accompanying it, by the spongy gums, and by the deep-seated effusions in the hams and elsewhere. On the other hand, amongst the poorer classes of the population one may overlook mild cases, where the symptoms mainly consist of the vague pains, with anæmia and ill-health, and the patients are likely to disregard a slight change in the gums, or a few spots on the skin. An inquiry into the diet, or the administration of lime-juice or vegetables, will soon determine the nature of the illness.

Treatment.—This is essentially dietetic; the important point being the use of abundance of fresh vegetable food. Thus the patient should have a liberal supply of mashed potatoes, cabbage, greens, or salad, and he should also have some fresh meat once a day. When the mouth is sore, and the patient is unable to chew, milk, beef-tea, mutton broth, and eggs may be given, and in any case, lime-juice or lemon-juice, to the extent of 3 or 4 ounces daily, diluted with water, and sweetened to taste. Patients who are very ill must be carefully nursed, and kept in the recumbent position, and they may require brandy if the circulation is feeble. Any drugs that are employed are required for local conditions, and have no influence over the general illness. The gums are benefited by the local application of nitrate of silver in stick, or by alum, Condy's fluid, decoction of oak bark, and potassium chlorate and chlorine water gargles. Diarrhœa, if present, may be met by bismuth or opium. For pain in the limbs, chloroform liniment may be used, and deep effusions have been treated successfully by friction with soap and water, and the internal use of potassium iodide.

For the treatment of infantile scurvy, the same principles must be followed. Fresh milk boiled, not humanised or sterilised, potato well steamed and beaten up with boiled milk, raw meat juice, grape juice, orange juice, and lemon juice, are the foods which should be employed.

Prevention.—For the prevention of scurvy in time of war, or on board ship, or at stations where vegetables are scarce, Parkes recommended, besides the use as far as possible of fresh and dried vegetables, that one ounce of good lemon-juice should be taken daily by each individual; that vinegar (½ oz. to 1 oz. daily) should be issued with the rations, and used in the cooking; and that citrates, tartrates, malates, and lactates of potassium should be issued in bulk and used as drinks, or added to the food (in soups and stews, or as salt). In the mercantile marine it is directed that an ounce of lime-juice should be served out daily to each man ten days after being at sea; the lime-juice is mixed, previous to being shipped, with 10 per cent. of brandy, rum, gin, whisky, or Hollands.

HÆMOGLOBINURIA.

Hæmoglobinuria (formerly called *hæmatinuria*) means the presence in the urine of the colouring matter of the blood, hæmoglobin, as distinguished from *hæmaturia*, the presence of blood itself, or, at least, the blood-corpuscles. Hæmoglobinuria arises when blood-corpuscles are broken up in the blood-vessels, so that hæmoglobin escapes free into the plasma, and is excreted by the kidneys. The plasma then becomes tinged of a pink colour, and for this condition the very clumsy name *hæmoglobinæmia* has been used. The excretion by the kidney possibly does not occur if the destruction is confined to the blood in the portal circulation (*see* p. 753). A partial destruction of the corpuscles takes place under several circumstances:—(1) The action of certain poisons, such as chlorate of potassium in large doses, pyrogallic acid, arseniuretted hydrogen, and naphthol. (2) The transfusion into one mammal of the blood of another; each kind of corpuscle becomes destroyed, and the serum is stained with hæmoglobin. (3) Exposure of the skin to extremes of temperature, such as burns or frost-bite. (4) The action of some fevers, so that a moderate degree of hæmoglobinæmia may result from scarlet fever or typhoid fever. (5) The paroxysmal form of hæmoglobinuria, in which the altered condition of the serum has been also demonstrated. Under all these circumstances the plasma has a reddish colour, and the blood-corpuscles have little tendency to form rouleaux. Very pale corpuscles (*shadow corpuscles*) are seen, and the hæmoglobin is deficient.

In hæmoglobinuria the urine is red, dark-red, or reddish-brown; it is acid in reaction, and deposits a dirty-brown sediment, consisting of epithelium, pigmented *débris* of corpuscles, perhaps casts containing blood-pigment, darkly stained urates, and opaque red granules of hæmoglobin. No blood-corpuscles can be seen. When examined by the spectroscope, the urine gives the two bands in the green and yellow characteristic of oxyhæmoglobin, and frequently another band nearer the red end of the spectrum, which is due to methæmoglobin. The urine is always albuminous.

PAROXYSMAL HÆMOGLOBINURIA.

This is a comparatively rare complaint, which consists in a liability to isolated attacks of hæmoglobinuria.

Ætiology.—It occurs in young adults, and middle-aged people up to fifty years of age, and is much more common in males than in females. In a large proportion of cases there is a history of malarial poisoning; in a certain proportion a history of syphilis;

and rheumatism is said to be an occasional antecedent. Hereditary tendency does not play a prominent part. The most common antecedent of an attack or paroxysm is exposure to cold, as from going out insufficiently clothed on a winter's day, or driving or walking far in the cold, or bathing in cold water. Exertion is another exciting cause.

Symptoms.—The beginning of the attack is marked in different cases by languor and weariness, a disposition to yawn, chill or rigor, pains in the limbs, nausea, vomiting, diarrhœa, and abdominal pain. Sometimes the fingers become white and cold, or the fingers, tips of the nose, and edges of the ears may become cold, livid, and even slough, constituting a condition of symmetrical gangrene (*see* Raynaud's Disease).

Towards the end of the attack an icteric tinge of the skin may occur, and urticaria has also been observed. The temperature may rise at the commencement, but soon subsides; and the whole duration of these symptoms is only from two to twelve hours. Either immediately after the first symptom, or only after three or four hours, the blood-coloured urine is passed, having the characters already described. But even this condition is only of short duration: in a few hours more the urine may be perfectly clear, and free from albumen and hæmoglobin; and in the intervals between the attacks it is always perfectly normal. Fagge pointed out that in some subjects of hæmoglobinuria, slighter chills may be followed by transitory albuminuria, which he explains by supposing that only a small number of corpuscles have been disintegrated, and that the hæmoglobin therefrom breaks up into hæmatin and globulin, of which the latter is excreted in the urine.

Paroxysmal hæmoglobinuria is not in itself dangerous; but the presence of renal cells and casts in the urine indicates that a nephritis is set up by the passage of the hæmoglobin; and in experiments with toxic agents producing hæmoglobinuria, the kidneys are found to be of a dark chocolate colour, from masses of the pigment collecting in the straight and convoluted tubes and in the glomeruli.

The disease may last several years, and if the attacks are frequent the patient may become anæmic, and acquire a sallow or faintly icteric tinge. Fagge records a case of chronic nephritis supervening upon paroxysmal hæmoglobinuria; but this may have been a coincidence.

Pathology.—There is no doubt that in this disease the corpuscles are broken up by the direct influence of cold upon them; and this effect may be produced locally—as, for instance, in the fingers. Many corpuscles are destroyed, the hæmoglobin escapes, and passes into the urine.

Treatment.—Direct exposure to cold must be carefully and systematically avoided, by the use of warm clothing, residence in warm rooms, and protection from night air as far as possible. It is much more difficult to diminish the susceptibility to its influence, which practically constitutes the disease. If there is any reason to suppose that syphilis is a predisposing condition, mercurials or iodides should be employed; more often malaria is credited with this, and quinine and arsenic are generally recommended. Their use must, of course, be continued for some time, and they may be given in daily doses of 5 or 6 grains of quinine, and 10 or 12 minims of the liquor arsenicalis. During the attack the patient should be made thoroughly warm by going to bed; and probably any renal irritation will be lessened by taking large quantities of fluid.

HÆMOPHILIA.

Hæmophilia, or the *hæmorrhagic diathesis*, is a disease characterised by a tendency to excessive or uncontrollable bleeding, either spontaneous or traumatic. It is congenital, and very often hereditary, so that the subjects of the disease, often known as "bleeders," are the children of bleeders, and their brothers or sisters suffer perhaps from the same malady. Though it is most frequent and most severe in males, it is transmitted through the female, who may herself be entirely free from it; in this it resembles pseudo-hypertrophic muscular paralysis. Beyond this hereditary transmission no other cause is known.

Symptoms.—These generally appear within the first year of life, though they are sometimes delayed till the seventh or eighth year. In the most severe degree, spontaneous hæmorrhages occur from the nose, the gums, and the mouth, and less commonly from the stomach, the lungs, or the genitalia; they are sometimes preceded by a feeling of fulness. Bleeding from the nose is the most common and also the most fatal. Alarming and even fatal hæmorrhages may also occur after the most trivial operation, such as lancing the gums, vaccination, the extraction of a tooth, incision of an abscess, and the application of leeches, or after accidental wounds or a cut finger. In all these cases there is the greatest possible difficulty in stopping the flow of blood. Besides these losses, hæmorrhage takes place readily under the skin from slight blows, or even spontaneously, producing bruises or blood-tumours.

In the intervals between the bleedings the subjects of hæmophilia may appear to be in perfectly good health, but the enormous quantity of blood which is sometimes lost may cause a high degree of anæmia, which lasts for many months. Hæmorrhage also takes place into the synovial cavity of the joints, especially the knee-joints: this occurs most commonly between the ages of seven and fourteen, and results from blows, or from exposure to cold or to damp. The swelling and pain closely resemble those of rheumatism or synovitis, for which indeed the symptom has been mistaken. This joint affection is accompanied with fever; it may recover, but returns again and again. Eventually the joint may become ankylosed. A rheumatic affection of the muscles, and the occurrence of trigeminal neuralgia, are described as occasional complications of hæmophilia.

Wickham Legg described three degrees of the disease: one, common in men, and scarcely ever seen in women, in which there is a tendency to every kind of hæmorrhage, spontaneous or traumatic, interstitial or superficial; a second, in which spontaneous hæmorrhages from the mucous membranes only are present; and a third, which shows itself only by spontaneous ecchymoses, and which occurs amongst the women of bleeder families.

The majority of the subjects of hæmophilia die from loss of blood before they are eight years of age; and though the chances of survival are greater after this period, even in middle age death may occur in the same way.

Pathology.—It would seem that the extraordinary conditions of hæmophilia must depend either on the state of the vessels or of the blood. But it has not been shown that the coagulability of the blood is really less, and there is no appreciable change in its saline constituents, in its albuminoids, in its fibrinogen, or in its structural elements. Nor has the microscope revealed much alteration in the vessels; it has been noticed sometimes that the arteries were unusually thin. It should be stated that the bleeding takes place from the capillaries as a general oozing, and the blood is more often venous than arterial in colour. The fatty degeneration of the heart and of the interior of the arteries found in some cases are probably the result of anæmia.

Diagnosis.—It must be remembered that women sometimes suffer after puberty from a hæmorrhagic tendency, shown by ready bruising, menorrhagia, &c., who have never been bleeders in early life, are not descended from bleeder families, and do not transmit the tendency to their offspring. In the absence of exact information as to the nature of true congenital hæmophilia, the relation to it of these cases must remain doubtful.

Treatment.—Patients who are the subject of hæmophilia should live on a light, unstimulating diet, and should pay particular attention to the bowels, any tendency to constipation being met by a laxative from time to time. The influence of drugs in ameliorating the condition is doubtful, but the tincture of the perchloride of iron is recommended by some. It is most important always to bear in mind the liability to bleed from any breach of surface; and the extraction of teeth, and all operations, large or small, should be avoided, unless absolutely necessary. If bleeding takes place, the various styptics must be employed in suitable doses, such as the perchloride of iron, ergot or ergotin, and gallic acid. The perchloride of iron may also be applied locally in solution, and in the case of bleeding after the extraction of a tooth, the crystals of this salt may be used to plug the cavity. As a last resort transfusion of human blood may be performed. Considering the serious nature of this disease, and the manner in which transmission takes place through the female sex, it is clear that women who belong to bleeder families, even though themselves not the subject of hæmophilia, should be forbidden to marry.

DISEASES OF THE URINARY ORGANS.

VARIATIONS IN THE COMPOSITION OF THE URINE.

Before proceeding to describe the diseases of the kidney, it will be best to refer to the variations in the constituents of the urine, and the methods of estimating some of them. Many of these variations are independent of disease in the urinary organs.

QUANTITY.

This is on an average fifty ounces, or 1450 cubic centimetres, per diem. It depends directly upon the pressure in the renal glomerulus, and consequently the urine is increased by conditions that raise the arterial tension either generally or in the kidney, and diminished by those that reduce this tension. Excessive secretion of urine is called *polyuria*, its suppression *anuria*. Daily causes of increase are the ingestion of fluids, and exposure of the bodily surface to cold; while it is diminished by abstinence from drink, by free sweating, and the loss of fluid from the circulatory system. The flow of urine is increased in the earlier stages of chronic granular kidney, in lardaceous disease of the kidney, in rare cases of cerebral disease, in diabetes insipidus, and in diabetes mellitus. A temporary increase is seen in hysterical attacks, as a result of mere nervous excitement (not infrequent during medical examination for life insurance), and from the administration of substances having a diuretic action, such as the acetates, citrates, and tartrates, and perhaps, most commonly, alcoholic drinks. The urine is scanty or suppressed in acute nephritis, in the last stages of chronic nephritis, in obstruction of the ureter unless the other kidney is equal to the task of secreting twice its normal amount, in all febrile processes, in cardiac failure, and after repeated vomiting, or profuse diarrhœa.

In order to ascertain the daily excretion of urine, all that is passed during twenty-four hours should be measured, and the mean of several consecutive days should be taken, so as to exclude the variations that are noticed under all circumstances.

SPECIFIC GRAVITY.

This varies directly as the amount of solids in the urine, inversely as the quantity of water—that is, of the urine itself. The excretion of both water and solids varies with the period of the day, and it is therefore desirable, for accurate estimation, to mix the whole of the urine secreted during twenty-four hours, and then to ascertain the specific gravity of the combined liquids. This is done by floating in the urine a *urinometer*, a glass instrument weighted by mercury and graduated from 0 to 50. In distilled water it sinks, so that the level of the liquid corresponds to zero; the specific gravity is read as 1000. In different urines it floats at higher levels. If the upper level of the liquid touches 15 or 20 it is described as having a specific gravity of 1015 or 1020 respectively. If the quantity of urine is too small to float a urinometer it may be diluted with one, two, or three volumes of water, as may be required, and the specific gravity will be obtained by multiplying the last two figures by the number which represents the dilution. Thus, a specimen of urine which, diluted with three volumes of water, gives a specific gravity of 1007, has really a specific gravity of 1028 ($7 \times 4 = 28$). Dr. G. Oliver has had made a graduated tube, and a glass bead which sinks in urine of 1008; if it floats the urine is gradually diluted until the bead sinks, when the mark on the tube reached by the fluid shows the original specific gravity. For rough purposes glass beads can be obtained which will sink respectively in urines of specific gravity 1005, 1010, 1015, 1020, 1025, and 1030.

The urine in health has a specific gravity of 1015 to 1025. It is diminished by most of the causes of polyuria, so as to be 1010, 1006, 1004, or less; as in chronic granular kidney, lardaceous disease, diabetes insipidus, hysteria, nervous excitement, and under the influence of diuretics. It is increased by all the causes of scanty urine, such as heart-disease, acute nephritis, and profuse sweating, and by conditions which increase the solids actually or relatively to the fluid secretion. In diabetes mellitus the specific gravity may be increased to 1030, 1040, or 1050, although the quantity of urine is many times more than the normal; the unnatural secretion of large quantities of sugar, while urea is often above the normal, accounts for this exceptional condition. Albumen occurs in urines of both low and high specific gravity; it must help in increasing the density, but it is, as a rule, in very small quantity when the conditions of the kidney allow a free secretion of urine.

SOLIDS.

The daily average of solids amounts to 950 grains, or 58 grammes. Their amount is accurately estimated by evaporating a specimen of the urine (collected during twenty-four hours), and weighing the residue. Since the specific gravity is in proportion to the solids, it ought to be possible to calculate the latter from the former with some degree of correctness. The average yield of solids on evaporation is about 4 per cent.; the average specific gravity is 1020. If the last two digits, 20, are multiplied by 2 we get 40—that is, the amount per 1000, and this corresponds to 4 per cent. Hence the rule to multiply the last two digits of the specific gravity by 2, in order to obtain the amount of solids per 1000; they must then be multiplied into the figure representing the quantity of urine, and divided by 1000; thus:—

$$\text{(Daily amount in cub. cent.) } \frac{1450 \times 40\ (= 20 \times 2)}{1000} = 58 \text{ grammes.}$$

The figure 2 thus employed is called *Trapp's co-efficient*, but the figure 2·33—*Haeser's co-efficient*—is thought to be more accurate, especially for specific gravities over 1025. The constituents of the urine whose increase is likely to affect the total daily solids are urea; the chlorides, phosphates, and sulphates of sodium, potassium, ammonium, calcium, and magnesium; urates of sodium, ammonium, and calcium; and the abnormal constituents—sugar and albumen. A great increase in solids is found in pyrexia. Excess of .the nitrogenous constituents (urea) is called *azoturia*; the presence of sugar and albumen respectively *glycosuria* and *albuminuria*. There is an actual diminution of solids in the conditions known as *renal inadequacy* and *anazoturia*.

The following require further consideration:—

Urea.—This constitutes about one-half the total solids of the urine, and is the chief form in which the nitrogen of the body is excreted. Its presence in the urine can be shown by evaporating an ounce of urine to about one-third its bulk, and adding a drachm of strong nitric acid: a flaky or feathery mass of crystals of nitrate of urea will be deposited as the mixture cools.

A rough estimate of the quantity of urea in any specimen may be sometimes given by nitric acid. In normal urine this acid gives no precipitate of nitrate of urea, unless after evaporation; but in highly concentrated urines, the salt crystallises out as the urine cools.

Quantitative Estimation.—A convenient method of estimating the urea is to decompose it by the addition of *sodium hypobromite* (or *hypochlorite*) into carbonic acid, water, and nitrogen—

$$(CON_2H_4 + 3NaBrO = 3NaBr + CO_2 + 2H_2O + 2N).$$

The nitrogen is measured, and from that the amount of urea can be calculated, the proportion of urea to its contained nitrogen being as 60 to 28, or 15 to 7. There are several forms of apparatus (Russell and West, Southall, Granville), in all of which a measured quantity of urine (5 c.c.) is mixed with a quantity of the hypobromite solution in such a way that the nitrogen gas, which immediately comes off, may be collected and measured in a graduated glass tube. The other results of the decomposition remain in solution. The graduations may represent the actual volume of nitrogen. In the apparatus generally employed the graduations represent not the volume of nitrogen gas, but the percentage of urea in the urine to which such volume is equivalent, and a tedious calculation is thus saved. The hypobromite solution rather readily undergoes decomposition after keeping, and it is better to make it afresh for each analysis.

In *Squibb's process* the liquor sodæ chloratæ of the U.S. Pharmacopœia, containing sodium hypochlorite, is used, and the nitrogen displaces water from a bottle completely filled and connected by a tube with the vessel in which the decomposition takes place. The displaced water has the same bulk as the liberated nitrogen, and the calculation of the urea can be made therefrom.

The average daily excretion of urea is 512 grains, or 33·2 grammes; it forms normally about 2 per cent. of the urine. The quantity varies very much under different circumstances. It is influenced most by the amount of nitrogenous food taken, being increased in proportion to this. It is increased also by the ingestion of water, and of table or other salts, and by the addition of fat to nitrogenous diet; but the further addition of farinaceous food again diminishes it. Moderate muscular exercise does not affect it.

In relation to disease, one must not rely only upon the percentage, but upon the absolute daily amount as far as that can be ascertained. The percentage may be high in consequence of diminished passage of water, as after sweating, or restricted supply of water, while the daily excretion of urea is normal. An increase of urea takes place in fevers, and in diabetes. A considerable decrease is present in all forms of Bright's disease, but also in other diseases, in which the amount of food ingested is much less than normal.

Chlorides.—Their presence is shown by a white precipitate on the addition of nitrate of silver to the urine, acidulated with nitric acid. Some idea of their quality may also be obtained in this way. Normally, the precipitate is thick and curdy: with diminished chlorides it may be a mere turbidity. The chlorides are lessened in all acute febrile processes, and especially in pneumonia, where they may be entirely absent.

Sulphates.—The daily excretion of sulphates is from 1·5 to 3 grammes. Of these the greater part are alkaline sulphates, precipitable by barium chloride; but from 5 to 10 per cent. are aromatic or ethereal sulphates, not precipitable by barium chloride, but convertible into the alkaline sulphates by boiling with hydrochloric acid. The ethereal sulphates are partly the result of putrefaction in the intestines, and are largely increased in disorders of the intestines (obstruction, peritonitis, constipation) leading to such changes.

Phosphates.—Phosphoric acid in the urine is combined with the alkaline bases, sodium and potassium, and with the earthy bases, calcium and magnesium. The alkaline phosphates form about two-thirds of the whole, and are always held in solution; the earthy phosphates form about one-third, and are spontaneously precipitated from the urine under various circumstances, as phosphatic deposits and calculi.

One of the causes of such deposition is an alkaline condition of the urine. The earthy phosphates are dissolved by acids, and again precipitated by alkalies. If the urine becomes alkaline in the body, or after it has been passed, phosphates are deposited; and the kind of phosphate depends upon whether the alkalinity is due to fixed alkali or to ammonia. The urine in health may be temporarily alkaline from the ingestion of much vegetable matter or other food containing citrates, tartrates, or malates of potassium and sodium. These are converted into carbonates in the intestines, and absorbed as such into the blood, and hence diminish the acidity of the urine, or render it alkaline.

Occasionally the urine, when passed, is quite turbid from a white deposit, consisting mainly of amorphous phosphate of calcium ($Ca_3P_2O_8$). This settles as a bright white deposit, immediately soluble in dilute acetic or nitric acid. Rarely there is mixed with this a crystalline phosphate ($CaHPO_4 + 2H_2O$), showing under the microscope rods and needles, lying loose or grouped into rosettes, stars, fans, and sheaf-like bundles, or club-shaped or bottle-shaped masses. This is called sometimes stellar phosphate. A magnesian phosphate ($Mg_3P_2O_8$), in elongated plates with oblique ends, has also been identified.

A much more common occurrence is this, that on heating a feebly acid or neutral urine a turbidity or thick white deposit of earthy phosphates occurs, which closely resembles albumen, but is at once distinguished from it by being dissolved by a drop of acetic or nitric acid. This effect of heat has been attributed to the driving off of carbonic acid, to the decomposition by heat of a combination between alkaline and calcium phosphates (Salkowski), and to the decomposition of a dicalcic phosphate ($Ca_2H_2P_2O_8$), held in solution by other salts, into a monocalcic phosphate ($CaH_4P_2O_8$) and the tricalcic phosphate ($Ca_3P_2O_8$), which is insoluble (W. Smith).

The addition of liquor potassæ or liquor sodæ to the normal urine at once throws down the phosphates.

If the urine becomes alkaline from the presence of ammonia, the ammonio-magnesian (wrongly called triple) phosphate is precipitated ($MgNH_4PO_4 + 6H_2O$). It forms triangular prisms, with bevelled ends, often very perfect indeed, but sometimes modified by the edges or angles being, as it were, planed off, or the ends or surfaces hollowed out. They often reach a large size, and are strongly refracting. This deposit may be mixed with that of the phosphate of calcium.

Calcium Oxalate.—The quantity of calcium oxalate secreted daily is very small, but it is sometimes seen as a deposit in the urine, or it forms calculi in the kidney. In urine containing an oxalate deposit there is mostly a pale grey mucus-like sediment, and above this a white dense layer, with a wavy, sharply-defined surface. Under the microscope will be found the minute octahedra of calcium oxalate, looking often like square envelopes, and measuring from $\frac{1}{5600}$ to $\frac{1}{750}$ inch in the side. In different positions they may seem to have a rhombic or hexagonal outline, and if the edges are not developed they may form square prisms, with pyramidal ends. A not uncommon variety is that of the "dumb-bell," which is really a disc, with a central depression on either face, lying on its side, and seen edgeways. Such formations no doubt result from slow precipitation in the presence of colloid matter, as shown by Ord.

Calcium oxalate crystals are with difficulty soluble in hydrochloric acid, and insoluble in acetic acid. Their deposition is sometimes due to excessive ingestion of vegetables (cabbage, rhubarb) containing oxalates; or to changes in the urine after secretion; or to disturbances of digestion. Their connection with certain symptoms, such as languor, depression, and hypochondriacal feelings (the so-called *oxalic acid diathesis*) is very doubtful, unless both the oxalate deposit and the symptoms are the result of indigestion.

Uric Acid and Urates.—The daily excretion of uric acid is only a few grains (8 to 15), so that the proportion in the urine is very small; nevertheless, deposits of the free acid and of its salts are not infrequent. As in the case of the other constituents, this precipitation does not prove that the quantity actually formed is in excess.

Uric acid is generally precipitated in very acid urine. Its deposits are distinguished by their yellow, orange, or red colour, and consist of minute shining grains, which with a lens or even the naked eye can be seen to have a crystalline, acicular, or prismatic shape. Under the microscope they show themselves as fusiform or lozenge-shaped crystals, with sharp ends and rounded sides; or as shorter and thicker crystals, with blunt extremities and more barrel-shaped; but generally in some modification of the diamond shape. They are frequently grouped together in radiating clumps or star-like masses. Thick crystals lying on their edges may seem to be rectangular, and the lozenge shape may not be detected till they roll over. Sometimes the sides of such masses are marked by a division into several plates (*striated*). These differences of shape are due to the conditions of the medium in which precipitation takes place. Their colour is that of the urinary pigment, for which they have great affinity. The crystals are insoluble in acids, but can be dissolved in alkalies.

Urates are, as a rule, precipitated, in an amorphous form, as a thick pink or red sediment (*brick-dust* or *lateritious*), which comes down as the urine cools, in cases where the quantity of water is deficient in consequence of free perspiration, febrile reaction, the low arterial tension of mitral disease and allied conditions. The colour is also due to a urinary pigment (*uroerythrin*), but it may be absent or amount to nothing more than a yellow tinge. A gentle heat will at once dissolve the deposit—for instance, the addition of hot water to the vessel containing it; and if the urine is being tested for albumen, the urates dissolve, and leave the fluid clear before the albumen begins to appear. This deposit consists chemically of the urates of sodium, potassium, ammonium, and calcium. They are strongly acid salts (formula MHU,M_2U) or *quadrurates;* and they are decomposed in the presence of water into free uric acid and *biurates* (MHU). Only under certain pathological conditions are biurates spontaneously deposited. *Ammonium biurate* occurs in ammoniacal urines in the form of dumb-bells or spheres, and the deposit is generally pale: *sodium biurate* constitutes the well-known acicular crystals found in gouty joints. *Neutral urates* (M_2U) do not occur in the animal body.

The Murexide Test.—If a particle of uric acid or a urate is heated on porcelain or a glass slide with a drop of nitric acid, and a drop of ammonia and water is added to the dry residue, a purple-red colour is developed. Liquor potassæ changes this to purple-blue.

The *quantitative estimation* of uric acid in the urine presents many difficulties, and is not suited for ordinary clinical work. A rough method is to add to a measured quantity of the urine about one-twentieth of its bulk of strong hydrochloric acid. The uric acid precipitated after twenty-four hours is collected, dried, and weighed; but the separation of the uric acid is incomplete, and the estimate is consequently too low.

Uric acid is probably derived from the disintegration of nucleoproteids in the body; and its excretion is most abundant about three hours after a meal. Its relation to gout will be discussed later; but it is excreted in very large quantities in some diseases, *e.g.*, leuchæmia.

Urinary Pigments.—The urine varies in colour both in health and disease. Vogel has prepared and published a series of tints, varying from a very pale tint of yellow, to red, red-brown, and almost black, and specimens of urine may in particular cases be referred to these for comparison. But, as a rule, it is sufficient to distinguish between pale urines, normal-coloured urines, and high-coloured urines in health; while in disease we may observe, in addition, different shades of red, reddish-brown, and brownish-black from the admixture of blood or bile-pigment, and an opaque white colour in chyluria.

The difference of tint in pale and high-coloured urines is mostly dependent upon the amount of water contained in them. *Pale* urines are of low density, and contain a large quantity of water and a small percentage of solids. They result from all causes which increase the flow of urine, such as free ingestion of fluids; a check to the cutaneous transpiration, as from cold; increased arterial pressure, as in early chronic Bright's disease; and nervous conditions, as hysteria, nervous excitement, and diabetes insipidus. The urine of diabetes mellitus forms an exception in being pale and abundant, while it has a very high density from the quantity of sugar contained in it. *High-coloured* urines are commonly of great density, scanty in quantity, and contain a large percentage of solids. They occur after profuse sweating, or diarrhœa; in fevers; and in disorders of the circulation, by which the flow through the glomeruli is diminished.

There are several pigments in the urine. Probably its colour is chiefly due, as shown by A. E. Garrod, to the substance previously described by Thudichum under the name *urochrome*. This pigment gives no spectrum. *Urobilin* exists in the normal urine in only small quantities; it gives a definite spectrum, with an absorption band at Frauenhofer's line F, which is darkened by the addition of acids to the urine, and caused to disappear by caustic soda or potash. *Hæmatoporphyrin* is derived from hæmatin; it occurs in minute quantities both in health and in disease, and more abundantly in the urine of rheumatic fever and some other disorders. It may not cause any appreciable difference in tint, but the urine has sometimes been dark red, as in some instances, after the administration of sulphonal. *Uroerythrin* is another pigment which gives the colour to pink urates. Uric acid deposits are coloured by urochrome, and also sometimes by uroerythrin (Garrod). It appears to be probable that the substance described as *febrile* or *pathological urabilin* is really the same as the urobilin already mentioned, and as such identical with *hydro-bilirubin*, and possibly *stercobilin*.

The urine may also contain *chromogens*—that is, bodies which do not at the time colour the urine, but develop a colour, either on standing, or on the addition of oxidising agents. The following are known:—(1) The chromogen of febro-urobilin, shown to exist in febrile urine, by the addition of nitric acid; (2) a chromogen found sometimes in the urine of anæmia, which, though quite pale when passed, may yield a deep red colour on the addition of nitric acid; (3) the chromogen of *melanin*, a black pigment which is developed on exposure, or on the addition of nitric acid, in the urine of patients suffering from melanotic sarcoma, although it is clear when passed; (4) indican, or the chromogen of indigo-blue. This is the result of the absorption from the intestinal canal of indol, which is formed from proteid decomposition. It exists in normal urine to a very small extent, but is greatly increased in all conditions leading to retention of intestinal contents, such as constipation, intestinal obstruction, and peritonitis. Its presence is detected by the addition of an equal quantity of hydrochloric acid, and then a few drops of a saturated solution of chloride of lime. Indigo is thus formed, and colours the mixture blue or violet. It can be separated by shaking with chloroform, which then forms a blue layer at the bottom of the test-tube. Sometimes the addition of nitric acid alone develops a blue, violet, or blackish colour, due to the separation of indigo.

Several *medicinal substances* colour the urine, or give colour-reactions with tests employed for other purposes. Rhubarb, which contains chrysophanic acid, makes the urine a deeper yellow; and santonin the same. The addition of an alkali will turn these urines red. Logwood gives a reddish tinge to the urine. Carbolic acid, taken internally, or absorbed from carbolic spray or dressings, often makes the urine dark-brown, or greenish-black, from the presence of oxidation-products, of which hydrochinone is one. Creosote may have the same effect. If potassium iodide or potassium bromide is being taken, nitric acid may darken the urine from the liberation of free iodine or bromine. Free iodine or bromine can be separated by shaking with chloroform.

Reaction of the Urine.

The urine is, as a rule, acid in reaction, and this acidity is probably due to the presence of the acid phosphate of sodium. After a meal the urine is often alkaline. The acidity of the urine during twenty-four hours is equivalent to about 14 grains of carbonate of sodium, or to 30 grains of oxalic acid. After it has passed from the body the reaction of the urine generally undergoes a change, becoming first more acid, then again less acid, and finally alkaline. These changes are due to fermentation from the presence of micro-organisms; for if urine is properly protected from contact with such bodies, it may be preserved for years. The increased acidity is due to more acid phosphates, as well as lactic and acetic acids. The alkalinity results from the decomposition of urea and the formation of carbonate of ammonium. In the former case uric acid is often deposited; in the latter, ammonio-magnesian phosphates, urate of ammonium, and phosphates and carbonate of calcium are thrown down.

It is, therefore, always desirable to know the reaction of the urine immediately it is passed from the body. If it is then alkaline, it must be ascertained whether the alkalinity is due to the volatile alkali, ammonia, or to fixed alkali (alkaline phosphate of sodium). A piece of red litmus paper turned blue by the urine, and subsequently gently warmed over a spirit lamp, will lose its blue colour in the case of ammonia; but will retain it in the case of the fixed alkalies. If ammonia is shown to be present, it is due to decomposition of the urine from retention in the bladder, or other parts of the urinary apparatus, as in pyelitis and pyonephrosis, precisely similar to what takes place in ordinary urine after it has been passed some time.

If the alkalinity is due to fixed alkalies, this results from changes in the blood, whether from diminished supply of acids, or increased supply of alkaline constituents. The most certain means of producing alkalinity of the urine is by the ingestion of large quantities of the potassium or sodium salt of citric, tartaric, acetic, or malic acid. These organic acids are decomposed in the intestines, and the alkalies are secreted in combination with the feeble acid, carbonic acid. The alkalinity which follows a meal is due partly to diversion of acid to the gastric juice, partly to the ingestion of alkaline citrates, &c., in vegetable food. Probably the latter has most influence, since, after a highly flesh or milk diet, the acidity is usually increased. The acidity of the urine is often diminished in melancholia, paralysis, anæmia, and chlorosis. It is increased in fevers and diabetes.

DISEASES OF THE KIDNEYS.

BRIGHT'S DISEASE AND NEPHRITIS.

GENERAL CONSIDERATIONS.

To Dr. Richard Bright belongs the credit of having first recognised the association of general dropsy and albuminous urine with a morbid condition of the kidneys. He found the kidneys sometimes large, pale, and smooth; at others, small, dark, and granular on the surface; and he regarded these two forms as different stages of the same process—namely, the deposit of a material which subsequently contracted. Later researches have shown that the changes in the kidney are for the most part of an inflammatory nature, being either acute, subacute, or chronic, and affecting the various tissues of the kidney with different degrees of intensity, but never of a kind to cause suppuration. Thus the term *Bright's Disease* is almost synonymous with non-suppurative inflammation of the kidney, though some writers would give it a wider, others a narrower, signification.

To understand the classifications that have been made, it is necessary to recognise the following structures in the kidney, which are liable to diseases, more or less independent of one another. They are:—(1) The tubules with their epithelium, forming the parenchyma of the kidney; (2) the interstitial tissue, very small in quantity in the healthy organ, but liable to considerable increase by inflammatory processes; (3) the blood-vessels, and the glomeruli, consisting of the vascular tuft, the capsule and the epithelial cells covering the former and lining the latter. Grainger Stewart's classification of Bright's disease was based upon this: he described a *tubal* nephritis, involving the epithelium of the tubules; an *interstitial* nephritis, affecting the connective tissue, and leading to fibroid contraction; and *waxy disease*, in which the arteries were first or mainly affected by the peculiar process of lardaceous degeneration.

By others the tubal form has been spoken of as *desquamative* or *parenchymatous;* the interstitial as *cirrhotic* or *granular;* the waxy as *lardaceous* or *depurative*. But it began to be discovered that in tubal nephritis the connective tissue did not escape, and conversely, that in the granular kidney the tubes were involved; and moreover, that the lardaceous change was accompanied by a certain degree of both tubal and interstitial change.

It is thus better to regard nephritis as in nearly every instance a diffuse inflammation, affecting all the several tissues at the same time, but under different circumstances involving the tubal epithelium, or the glomeruli, or the intertubular tissue most, so that the divisions into tubal and interstitial kinds may still be maintained to a certain extent; and to these a third form may perhaps be added—namely, *glomerular* nephritis. In addition, we must distinguish between *acute* and *chronic* lesions, and so a considerable number of forms would seem to occur. The following is the classification which I shall adopt in describing the several varieties of nephritis, with their accompanying clinical symptoms:—

1. *Acute nephritis*, tubal and glomerular.
2. *Chronic tubal nephritis*, meaning thereby a form in which the renal epithelium is at first mainly affected, though intertubular growth may subsequently take place and lead to contraction.

3. *Chronic interstitial nephritis*, resulting in the red granular kidney.

The position of lardaceous disease may be considered somewhat doubtful in relation to Bright's disease; it is primarily degenerative, and the inflammatory change is secondary. Clinically it presents many resemblances to the inflammatory forms, and some important differences. It has been usually included under Bright's disease. In the present volume I shall follow more recent writers, and consider it separately.

In addition to the above forms of primary nephritis constituting Bright's disease, two other varieties have to be described:—

4. *Consecutive nephritis*, chronic, acute, diffuse, and suppurative, the results of disease in the lower urinary passages.

5. *Metastatic suppurative nephritis.*

There are some symptoms which occur more or less in all forms of Bright's disease, and which will now be described. These are—1. Albuminuria. 2. Hæmaturia. 3. The presence of casts in the urine. 4. Dropsy. 5. Cardio-vascular changes. 6. Ocular changes. 7. Hæmorrhages. 8. Secondary inflammations, and other lesions. 9. Uræmia.

ALBUMINURIA.

The presence of albumen in the urine is the most constant sign of Bright's disease, and may be detected in various ways.

Heat.—If clear urine containing albumen be heated in a test-tube, it will become opaque from the precipitation of this substance. According to its quantity the precipitate will be a mere opalescence, a decided turbidity, or a thick creamy deposit; on the cooling of the urine it will separate into small particles or flakes, and gradually subside to the bottom of the test-tube. The best way of applying the test is to fill a test-tube to one-half or two-thirds of its length, and, holding it by its lower end, to warm the upper part of the urine. The heat is thus confined to that portion of the urine, and whatever slight change takes place in the clearness can be recognised by comparison with the cool urine below; whereas, if heat were applied to the bottom of the test-tube, it would reach, by convection, the whole of the urine at once, and a slight opalescence might, for want of comparison, be overlooked. The value of this method is shown in cases where an albuminous urine is turbid from a deposit of urates. Heat will at first dissolve the urates, and then precipitate the albumen. A long column of such urine may be heated in its upper two-thirds to clear it from urates, and then further heated in its upper third to throw down the albumen, when the three strata of urates, clear urine, and albumen may be compared with one another.

Some precautions are necessary. First, heat may precipitate substances that are not albumen. In certain states of the urine, a precipitate comes down which is to the eye indistinguishable from albumen; it is due to the earthy phosphates (*see* p. 774), and is at once dissolved by a drop of nitric acid, whereas a precipitate of albumen is unaffected or becomes denser. If the two occur together, there will only be a partial clearance on the addition of nitric acid. Heat also throws down a proteid substance, paraglobulin, but this probably always accompanies the serum-albumen of Bright's disease in greater or less quantity, and its significance as to disease of the kidney is practically the same.

Secondly, albumen, though present, may fail to be coagulated by heat. This occurs when the urine is alkaline, and the serum-albumen has been converted into alkali-albumen, which is not precipitated by heat; the fallacy can be guarded against by the addition of a drop or two of acetic acid to the urine, so as to render it faintly acid before boiling. On the other hand, it occasionally happens that serum-albumen is converted into acid-albumen or syntonin, which is similarly unaffected by heat. Owen Rees used to point out that this might happen if the urine to be boiled were placed in an unwashed test-tube containing a trace of nitric acid from a previous examination. In any case, the experiment must be performed upon a clear urine; if turbid from urates, a gentle heat clears it; if from phosphates, a drop or two of acetic acid should be added; if from any other deposit, the urine should be filtered.

Nitric Acid.—This precipitates serum-albumen, as well as alkali-albumen, and acid-albumen. If the albumen is in large quantity, a drop or two of strong nitric acid added to the urine will carry down a thick curdy white precipitate; but for smaller quantities, the test is best applied by placing a little nitric acid in the bottom of a test-tube, and very gently pouring the suspected urine down the side of the tube, so that it flows on to the surface of the acid without mixing with it. At the line of junction a layer of albumen forms, of white colour if abundant, a mere thin grey line when the quantity is very small. In the former case also it forms at once, in the latter it may take several seconds, or two or three minutes, or even half an hour, according to Roberts (*see* Quantitative Tests).

There are but few fallacies connected with this test. First, in urine containing an excess of urates, these are sometimes precipitated; they form a cloud which is generally suspended in the column of urine some distance above the nitric acid, instead of lying immediately upon it; and the application of a gentle heat will at once dissolve them. Secondly, the urine of persons who are taking copaiba internally contains a resinous acid, copaivic acid, in combination with bases. If nitric acid be added to this the resinous body is thrown down, generally as a cloud evenly diffused through the urine. This precipitate is also dissolved by heat. Thirdly, in many specimens of urine the addition of nitric acid gives rise after some time, when the mixture has become cold, to a crystalline deposit of nitrate of urea; but it has no resemblance to albumen, consisting, as it does, of lamellar crystals, radiating in various directions.

Picric Acid.—This also forms a very delicate test for albumen, and has been strongly advocated by Johnson. A test-tube should be more than half filled with urine, and a saturated solution of picric acid, which has a low specific gravity, should be poured on to it so that the liquids may mix as little as possible; at the line of junction a delicate white line, or a thicker white cloud, at once forms, which is not dissolved by heat. Besides the three forms of albumen, picric acid also precipitates urates, alkaloids, and peptones. All of these disappear at once on warming the urine. Quinine is the only alkaloid that is likely to be taken in sufficient quantity to be precipitated by picric acid. There seems to be some doubt as to the precipitation of mucin by this test. Johnson, at any rate, denies it.

Potassium Ferrocyanide.—This reagent throws down albumen in acid solutions. Citric acid is therefore used as well as the ferrocyanide. The two reagents may be used in solution or in the form of the "pellets" devised by Pavy, which may be easily carried in the pocket. Into the suspected urine a citric acid pellet is first dropped, and when that is dissolved, a pellet of the ferrocyanide; if any albumen is present it forms a cloud as the salt dissolves. Citric acid itself may precipitate urates and mucin; if the former are thrown down, a fresh specimen of the urine should be diluted with an equal quantity of water and tested again. If mucin is precipitated by the citric acid, this specimen should be left untouched, and to another specimen both citric acid and ferrocyanide should be added, when the difference between the two deposits will show the presence of albumen. The ferrocyanide does not precipitate peptones.

Potassio-iodide of Mercury.—This precipitant of albumen, like the ferrocyanide, requires an acid urine, and citric acid may again be used for this purpose. The salt throws down the three forms of albumen, but, like picric acid, it precipitates urates, alkaloids, and peptones.

Brine.—Sir W. Roberts introduced a saturated solution of common salt, acidulated with one per cent. of hydrochloric acid, as a test for albumen. It should be used in the same way as nitric acid, by placing about a drachm in the bottom of the test-tube, and pouring the urine gently on to it; the albumen is precipitated at the line of junction. It has the advantage, if carried as a liquid test, of not being corrosive like nitric acid; but the disadvantage of precipitating peptones.

The following are *quantitative tests* for albumen :—

Weight of Precipitate.—The most accurate method is to thoroughly precipitate by heat all the albumen from a definite quantity of urine, and then dry and weigh the precipitate. This is a more or less troublesome process, requiring an accurate chemical balance, and is not suitable for ordinary clinical purposes.

Measurement of Precipitate.—The urine is boiled in a long narrow test-tube, and the albumen is allowed to settle at the bottom. After complete subsidence the length of tube occupied by the albumen is compared with the whole length occupied by the urine; it may be one-tenth, one-third, one-half, or more. This gives no information as to the absolute amount of albumen in the urine, but it may show the daily variations of the albumen in any case, and whether the urine contains much or little of it. A very albuminous urine becomes almost solid on boiling; a large quantity settles down to one-half or one-third of the urine in the tube; a mere turbidity, on boiling, will form a little deposit at the bottom of the tube—that is, perhaps from a hundredth to a fiftieth part.

Esbach's Test.—In this the same principle is applied in a more accurate way. A test solution is prepared, consisting of one part of picric acid and two parts of air-dried citric acid in one hundred parts of water. A graduated tube from six to eight inches long and half an inch in diameter is filled up to a certain level (two and a quarter inches) with urine, and then for a certain distance (one and a half inches) with the picric solution. The precipitated albumen is allowed to settle for twenty-four hours, and then the space it occupies in the tube is compared with a series of marks which represent the number of grammes of albumen for each litre of urine; or, in other words, the amount of albumen per thousand of urine. Thus, for instance, if it reaches mark 3, there are three grammes of albumen for each litre of urine, or 3 per 1000, or ·3 per cent. From this, of course, if the daily amount of urine is known, the absolute quantity of albumen passed can be calculated. In some urines the deposit is increased by the presence of uric acid, and thus the albumen may be over-estimated. Johnson says this is obviated by omitting the citric acid and using a solution of picric acid of five grains to the ounce.

Roberts' Test.—This consists in a delicate and special application of the nitric acid test. Roberts found, when the amount of albumen was so small as to require from thirty to forty-five seconds for the thin ring of albumen to form at the junction of the acid and the urine, that it proved on careful chemical analysis to be equal to ·0148 grain per ounce, or ·0034 per cent As most specimens of urine contain more than this, so that the ring of albumen appears almost at once, the specimen must be diluted—by successive equal quantities of water—until the albumen is in sufficiently small quantity to require the time named. The amount of dilution must be noted, and the amount of albumen is obviously directly proportional to the number of times the urine has been diluted by its own quantity of water. Thus, if the urine has to be increased to fifty times its bulk to lessen the proportional quantity of albumen sufficiently, it must contain fifty times the amount of albumen specified—*i.e.*, 50 × ·0148 grain per ounce or 50 × ·0034 per cent. It will be seen that seventy dilutions represent about one grain per ounce, and 300 dilutions equal one per cent.

The Causes of Albuminuria.—In considering the reason why albumen appears in the urine in Bright's disease, we must remember that the occurrence of albuminuria is not limited to cases of nephritis, but accompanies a variety of other disorders. The different conditions under which albuminuria has been observed may be enumerated as follows:—

I. Arising in the kidney—
  1. Acute and chronic nephritis, and contracted kidney, forming Bright's diseases; consecutive nephritis and cystic kidney.
  2. Suppurative nephritis.
  3. Degenerative changes, such as lardaceous disease and tubercular kidney.
  4. Acute febrile processes, probably causing temporary degeneration of the renal cells.
  5. Venous obstruction in diseases of the heart and lungs, and local disturbances of the circulation.
  6. Malignant endocarditis and embolism of renal arteries.
  7. New growths and parasites.
  8. Temporary obstruction of the ureters.
  9. Nervous disorders, such as apoplexy, convulsions, and concussion.
  10. Chronic general disorders, like leuchæmia, diabetes, and anæmia.
  11. Disturbances of digestion, and disorders of a temporary nature, including so-called cyclic and physiological albuminuria (*see* Functional Albuminuria).
  12. The influence of certain poisons, and the presence in the blood of forms of albumen other than serum-albumen.

II. Arising in the urinary passages below the kidney—
  1. Disease of the pelvis of the kidney, calculous pyelitis, and tubercular disease.
  2. Tubercular disease of the ureter.
  3. Cystitis and tubercular disease of the bladder.

In this last group there is no difficulty in explaining the occurrence of albumen; it is not infrequently accompanied by blood, and may be the result of rupture of vessels, as in the case of calculus, or it is due to inflammatory and ulcerative processes by which an albuminous secretion is provided in the urinary passages, just as it would be on any other mucous surface or on the skin.

The cause of renal albuminuria has been the subject of much debate, but it is generally believed now that it depends upon damage to the renal epithelium; especially that lining the glomeruli. It will be seen that this explanation is more or less applicable to a great number of the conditions under which albuminuria arises.

In acute and chronic forms of nephritis glomerular changes are almost constant, and according to Ribbert they are always the earliest indications of inflammation. In febrile processes, the epithelium of the tubules has long been known to be affected with the condition known as cloudy swelling, and the still more delicate glomerular epithelium would almost certainly not escape. In the venous obstruction of heart-disease, it is quite likely that the epithelium suffers from the deficient supply of blood through the afferent arteries. This is supported by Nussbaum's experiment, in which a temporary ligature of the renal artery was followed by albuminuria on restoration of the arterial flow; presumably in the interval the glomerular epithelium was sufficiently damaged to be unable for a time to resist the passage of albumen. In nervous diseases there may be vasomotor disturbances which would act in like manner upon the glomerular circulation; and in chronic disorders the glomerular epithelium may share in the general depression of vitality.

But though this may be the mechanism of albuminuria in a great number of instances, it seems difficult to believe that at no time can it result from transudation directly into the tubes themselves; and this in two ways—(1) from extreme pressure of congestion; (2) as an inflammatory exudation. In so far as increased pressure, whether from general conditions of the circulation (exercise, full meals), or from the pressure of leucocytal infiltration in the substance of the kidney, leads to distension of the glomerulus, this cause of albuminuria falls within the scope of the more generally accepted theory. But it seems possible that some transudation may take place, for instance, from the vasa recta into adjacent tubules. Similarly, if in an acute inflammation the substance of the kidney is infiltrated with inflammatory serum, it is held by some (Greenfield) that this escape into the tubules may contribute albumen to the urine.

The presence of other proteids in the urine.—Besides serum-albumen, other proteids may be found in the urine. *Serum-globulin* (or *paraglobulin*) commonly accompanies albumen and is precipitated by cold nitric acid and picric acid. It is often in excess in the urine of lardaceous disease and may be detected by pouring the urine into a large bulk of distilled water, when the globulin is precipitated. *Albumoses* also occur in the urine, generally in association with albumen, but sometimes without; and they are due mostly to absorption from purulent effusions, to diseases of the liver, especially acute yellow atrophy, to ulceration of the bowel in typhoid fever or otherwise, to scurvy, and to toxic and puerperal conditions. They may occur in large amount without albumen in some cases of chronic Bright's disease (granular kidney). Albumoses may be detected by the gradual addition of nitric acid to urine in a test-tube, when a precipitate forms, and if this be heated it is dissolved, and again precipitated as the mixture cools. Albumoses also give the biuret reaction—a pink or rose-red colour when the urine is floated in a test-tube over a small quantity of Fehling's solution. If albumen is also present the colour will be mauve or purple. *Peptones* also occur in the urine, but have not much significance. No doubt some of the cases formerly called peptonuria, were really instances of albumosuria. Peptones give the biuret reaction. They are not precipitated by nitric acid, nor by saturation of the fluid with ammonium sulphate, which precipitates the other proteids. This and other salts may be used for the separation of the proteids, since ammonium sulphate precipitates all proteids, except peptones, sodium magnesium sulphate precipitates serum-albumen, and magnesium sulphate throws down serum-globulin.

HÆMATURIA.

In acute forms of nephritis the urine often contains blood or some of its constituents other than albumen. This is rarely in such quantity as occurs from calculus and growths in the kidney; but it sometimes gives the urine a bright-red colour, more often a dirty-brown colour and turbid appearance from the presence of methæmoglobin. Generally with this there is a granular reddish-brown sediment. The presence of blood, which in this last case may be doubtful, is determined in several ways.

The Microscope.—By this we can recognise blood-discs, in cases where there is no suspicion, from the colour of the urine, that blood is present; if the urine is distinctly red, or brown and turbid, the discs will be there in abundance. From suspension in the urine they may have lost their biconcave form, and are often shrivelled, and have crenate edges, or may present protrusions of their substance. They remain visible for the longest time in acid and dense urines, but may be quickly dissolved in ammoniacal urine, or in urine of low specific gravity.

The Spectroscope.—The spectrum of urine containing blood shows two absorption bands in the yellow and green portions between Frauenhofer's lines D and E, the darker broader band being nearer to D. These are due to oxyhæmoglobin. Methæmoglobin gives three absorption bands, two in very nearly the same position as those of oxyhæmoglobin, and a third about half-way between C and D.

Heller's Test.—This consists in adding caustic potash or soda to the urine, and then boiling. The phosphates which are precipitated are coloured pink or red by the hæmoglobin which they carry down with them.

The Guaiacum Test.—To the urine, in a test-tube, are added a few drops of tincture of guaiacum, and then about half a drachm of ozonic ether. Quickly or slowly, according to the quantity of blood, a blue colour forms at the junction of the fluids, and diffuses itself through the ether which floats on the surface; its appearance may be hastened by shaking the mixture. The test is not absolutely trustworthy.

It is often desired to know in what part of the urinary passages the hæmorrhage has arisen. In renal hæmorrhage the blood is often intimately mixed with the urine; in bleeding from the bladder it is more separate; in urethral hæmorrhage the blood comes apart from the urine. The diagnosis is also helped by any coagula that may form: in renal hæmorrhage blood casts (*see* p. 783) are found, in hæmorrhage from the ureter there may be long stringy coagula, and in cystic hæmorrhage large flat oval clots 1½ inches in length and 1 inch broad with fringed edges may be present.

CASTS.

These are solid bodies, which are detected by the microscope in the urine, and if sufficiently numerous, form a sediment visible to the naked eye. Where they are too few to be easily detected, they may be found after centrifugalising the urine. They are cylindrical in shape, and from ·01 to ·05 mm. in breadth; but they vary still more in their length, which may reach 1 or even 2 mm., so as to stretch right across the field of the microscope, but is more often from five to ten times the breadth. Their connection with the kidney is proved by finding them after death, *in situ*, in the renal tubules. The following varieties are distinguished.

Hyaline Casts.—These are transparent colourless cylinders, with refractive properties so like those of the fluid in which they lie, that they are discovered with great difficulty, unless they are stained by the addition of carmine or iodine, or one of the aniline dyes, such as gentian-violet. They are homogeneous, soft, and flexible, straight, or curved, and varying in length. Occasionally they have other deposits adherent to or embedded in them, such as red blood-corpuscles, leucocytes, epithelial cells, granular masses, fatty granular globules, crystals of urates or oxalates, or particles of hæmatoidin.

Some of the varieties of casts described below probably have the same hyaline material as a basis, which is then completely covered by, or mixed up with, the other elements.

Hyaline casts are often spoken of as "fibrinous," but it appears that they are neither pure fibrin nor pure albumen, but can at present only be described as albuminoid. Most probably they are produced by the coagulation of a fluid exuded by the glomeruli, as they form with great rapidity—sometimes in a few hours. But they are thought by some to be due to an altered condition of the renal epithelial cells, or of leucocytes.

They occur in the different forms of nephritis, and in the congestion produced by heart-disease; they are nearly always in association with albuminuria, but may precede for a few hours or days the appearance of the albumen, and may continue for a time after its disappearance. This, of course, does not apply to albuminuria dependent on lesions outside the kidney.

Granular Casts.—These are not so transparent as hyaline casts, being sometimes like ground-glass, sometimes darker and much more opaque.

Epithelial Casts.—These consist of cells of the renal epithelium held together by, or embedded in, the coagulable material which makes up the hyaline casts. The epithelial cells may be more or less distinct; and some have thought that they are more often swollen leucocytes, than actually the cells of the renal tubules.

Fatty Casts.—These are either hyaline casts in which globules and granules of fat are embedded, probably from the disintegration of epithelium in a state of fatty degeneration; or they actually consist of such fatty epithelial cells themselves.

Blood Casts.—These result from the coagulation of blood that has been effused into the renal tubules; they are easily recognised by their colour, and by the size and close aggregation of the blood-corpuscles composing them. Their presence in a case of hæmaturia proves that the blood comes from the kidney itself.

Lardaceous Casts.—Casts occasionally occur, which are stained of a reddish-brown colour by iodine, and seem to consist of lardaceous material. They do not, however, occur always in cases of lardaceous disease, nor are they confined to such cases; they are thought to result from some secondary change in previously hyaline casts.

Though the nature of the casts must depend to some extent on the condition of the kidney, conclusions on this point must not be too hastily drawn from them. Frequently two or more forms are found in the same urine, and we must be guided by the predominance of one or the other. Hyaline and granular casts are found in all forms of nephritis, acute or chronic. Blood casts and epithelial casts are most common in acute cases; fatty casts are most frequent in cases of chronic nephritis undergoing fatty degeneration.

Dropsy.

Two forms of dropsy may be recognised in renal disease.

One of these is identical with the dropsy which results from heart-disease, and is, indeed, due to the secondary disturbances of the heart which subacute or chronic Bright's disease induces. In this, the dropsy occupies especially the lower extremities and the lower half of the body, while the face, arms, and upper half of the body may be free.

The other form is the characteristic *renal dropsy*, which is seen typically in acute Bright's disease, and involves the whole surface of the body and the great serous cavities. Frequently the first change noticed is some puffiness of the eyelids when the patient rises in the morning. This may subside in the course of the day, but if the feet be examined at night there is œdema just below the ankles. There is, indeed, a small quantity of effusion into the subcutaneous tissue, which always seeks, by force of gravity, the most dependent part. During the daytime it reaches the feet; in the recumbent position of sleep it diffuses itself generally, but is most pronounced in the loose tissues of the eyelids. If the patient takes to his bed, it will leave both the face and the feet, and accumulate in the tissue over the sacrum, which has now become the most dependent part. In more advanced cases, the dropsy becomes general, and the skin of the whole body is œdematous. The face is full and rounded, the eyelids are distended, and almost close the eyes; the limbs become enlarged, shapeless, and remind one of bolsters; the trunk is enlarged; the loose skin of the penis and scrotum is so stretched that the prepuce looks like a bladder, and the scrotum may attain the size of a fœtal head. Wherever slight pressure is applied, as by the finger of the doctor, or by the bands, strings, or folds of clothing, it produces, by the displacement of the fluid, a deep impression, which is only slowly effaced by the return of the fluid. This is called *pitting on pressure*. Even then the influence of gravitation on the distribution of the dropsy may be seen, for if the patient lies for any length of time on one side, the arm of that side will become more swollen than the arm which is uppermost; and the same will happen with the two sides of the face. When this general dropsy, or *anasarca*, is present, there is, as a rule, some effusion into the peritoneal cavity (*ascites*), into one, or more frequently both sides of the chest (*hydrothorax*, dropsy of the pleura), and it may be into the pericardial sac. Of these, the first is perhaps most often recognised; while the pericardial and pleural effusions may be comparatively slight.

If incisions be made into the skin, or if one or more Southey's drainage tubes be inserted, a quantity of fluid will drain away, which may amount to eight or ten pints in a few hours, and the dropsical limbs will rapidly get smaller. The fluid is colourless, of low specific gravity (1007·65, Bartels), and contains a small quantity of albumen, inorganic salts, and urea. A feature which is constantly present in renal dropsy is a high degree of pallor—the lips are almost colourless, the cheeks are pallid, and the whole body has a waxy whiteness. This is partly due to the dilution of the blood and its poverty in red corpuscles; partly to the distension of the skin and subcutaneous tissues.

The typical anasarca of renal disease is by no means so easily explained as the dropsy of heart-disease. In this latter it is clear that over-filled vessels on the venous side of the heart are unable to take up the fluid, which naturally transudes into the subcutaneous tissue. In renal dropsy an analogous mechanical explanation is held by many. As a rule, much dropsy coincides with decided decrease in the quantity of urine secreted, and it is an obvious suggestion that the liquid which cannot pass out by the kidneys escapes into the subcutaneous tissue; and in cases that recover, it is common to observe that the dropsy diminishes coincidently with an increased flow of urine. Moreover, in chronic renal disease (granular kidney) the urine is at first abundant; and dropsy is slight or none until the urine becomes scanty from other renal changes, or until the heart becomes secondarily involved, and a cardiac dropsy results. As to the details, Ralfe supposes that the retention of water by the kidneys, while the patient drinks the same as in health, increases the arterial tension throughout the body, and this relieves itself by transudation into the tissues. But suppression of urine undoubtedly occurs in some other ways without producing anasarca, and in one form of dropsy urine is abundant.

The following are some other hypotheses which have been advanced to account for acute renal dropsy:—One, that the exudation is really of an inflammatory nature; another, that the specific gravity of the blood is lowered in renal disease, and hence transudation is facilitated—but the experimental production of hydræmia does not lead to dropsy; another, that there is a peculiar alteration of the vessels of the skin and subcutaneous tissue set up by the disturbance, whatever it is, that has caused the renal disease.

CARDIO-VASCULAR CHANGES.

These are, in acute cases—(1) High arterial tension, and (2) dilatation of the left ventricle; in chronic cases—(1) High arterial tension ; (2) thickening and atheromatous change in the arteries ; (3) hypertrophy and dilatation of the heart.

Arterial Tension.—In acute nephritis, quite early in the history of the case, the pulse will be found abnormally hard, resisting a sphygmographic pressure of four, six, or eight ounces, or more, and giving a tracing which is often highly characteristic, the tidal and the dicrotic wave being both very pronounced, and so forming an outline like a hill with three nearly equal peaks (*see* Fig. 22). Coincidently with this the cardiac sounds are modified ; the first is reduplicated or broken, and the second is accentuated, or ringing, over the base of the aorta. In some severe cases, after a certain duration, dilatation may be observed, the impulse being under the nipple or even outside it; and the heart may become irregular in its action. If the illness is much prolonged, there is a tendency to the hypertrophy which is seen in chronic cases.

High arterial tension is also the rule in chronic Bright's disease ; the pulse is hard and resists compression, and yields a tracing in which the tidal wave is high and long, giving a flat top to the outline, but in which the dicrotic wave is only slightly marked (*see* Fig. 22).

Changes in the Vessels.—Atheromatous changes take place in the artery walls, and are no doubt in part the result of the strain to which the vessels are put; but they are neither constant in, nor peculiar to, Bright's disease. Much more characteristic is the more or less uniform thickening (arterio-sclerosis) which affects the small arteries all over the body, as well as the vessels in the kidney itself. As to the nature of the thickening of the arteries, very different opinions have been expressed. Johnson described it as *hypertrophy* of the *muscular* coat; Gull and Sutton regarded it as a transformation of the coats into a *hyaline-fibroid* material (arterio-capillary fibrosis).

Changes in the Heart.—Hypertrophy of the heart is a very frequent change in chronic Bright's disease, and in granular kidney it reaches its greatest extent. It may show itself by the usual physical signs—namely, displacement, and heaving character of the impulse. The weight of the heart varies from seventeen to twenty-eight ounces, and the hypertrophy in the more marked cases affects the right as well as the left ventricle. In advanced cases it is accompanied by dilatation, and this has an important bearing upon the clinical features of the illness. With its occurrence, the circulation, already obstructed by the high arterial tension, falls more and more, the action of the ventricle becomes irregular, regurgitation takes place through a leaking mitral orifice, and eventually the complete picture of a primary valvular disease may be developed, with a mitral murmur, engorgement of the lungs and liver, ascites, and dropsy of the lower half of the body.

The cause of the cardiac hypertrophy in renal disease has been no less hotly debated than the many other conditions in this interesting disorder. The early view was that the blood, poisoned by urinary constituents, circulated with difficulty through the capillaries of the body, and the left ventricle necessarily hypertrophied to overcome the resistance thus created. Johnson, adapting the theory of a poisoned blood to his view of a hypertrophy of the muscular coat of the arteries, supposed that the vessels exerted a kind of stopcock action, and contracted—hence becoming hypertrophied—in order to prevent the poisoned blood reaching the tissues; and that the heart hypertrophied in consequence of this arterial resistance. Traube thought that the disease of the kidney so diminished the area of outlet for the blood from the aortic system, as to materially increase the tension in the rest of the body, hence the cardiac hypertrophy; but it appears that ligature of the renal artery does not raise the arterial pressure. Fagge in part followed Cohnheim, and thought that if a certain area of the kidney becomes functionless from disease, the same amount of blood as formerly must be forced through what remains of the kidney, in order to properly depurate the body; to do this the heart must hypertrophy, and since its force would be directed uniformly upon the whole systemic circulation, the arteries of the rest of the body are hypertrophied to protect the area to which each belongs from undue pressure. This is then a stopcock theory, in which the arteries protect from undue pressure, instead of from diseased blood. By some, chronic Bright's disease, in the form of granular kidney, is regarded as a general and simultaneous affection of the heart, the arteries, and kidneys; but if this were true, we still have to account for the precisely similar changes which occur in chronic parenchymatous nephritis, in which case the renal disorder undoubtedly precedes the other symptoms. Recently Bright's original view that the heart hypertrophied from the irritation of its cavities by the impure blood has been resuscitated.

Ocular Changes.

In chronic forms of Bright's disease, especially in granular kidney, and also in lardaceous disease, changes of much importance take place in the fundus of the eye. They consist of—(1) œdema of the retina, causing opacity and swelling; (2) white spots or patches in the retina; (3) hæmorrhage into the retina; (4) papillitis; and lastly (5) atrophy of the retina and papilla, the result of the preceding inflammation. These are generally grouped under the term *albuminuric retinitis*.

The most frequent and characteristic are the white spots, which are often of a glistening or silvery white colour, at first very small, though subsequently enlarging, partly by coalescence.

They are most numerous in the neighbourhood of the optic disc, and especially around the yellow spot, where they may have a more or less radiating arrangement. Often a large number are grouped together so closely as to resemble a piece of mosaic. They are due to a degenerative and fatty change in the elements of the retina, especially in the nerve-fibres, but also in the corpuscles and Müller's fibres. The nerve-fibres become thickened and swollen, and varicosities form, which are filled with fat-globules; compound granule-cells are also found in their deeper layers.

The hæmorrhages vary in size, are often small, sometimes elongated, pointed and divided, or "flame-shaped," and radiating from the optic disc as a centre. They are mostly seen in conjunction with the white spots.

Neuritis or papillitis is shown by the swelling of the disc, the opacity or blurring of its edge, the partial or complete concealment of the retinal vessels, especially the arteries, and the filling and tortuosity of the veins. The condition varies in different cases. Sometimes it is so great as to resemble the neuritis of marked cerebral disease. More often there is but slight prominence, but diffused opacity spreads far on to the surrounding retina. In many cases the papillitis, spots of degeneration and hæmorrhages co-exist; sometimes one, sometimes the other, being more pronounced.

Probably, in the majority of cases, the changes persist until the patient's death; but improvement may take place, neuritis subsiding, diffused blood being absorbed, and even the spots of degeneration disappearing. Loss of vision is more or less in proportion to the extent of the changes, and of the implication of the yellow spot. In the earlier stages there may be no appreciable loss, and total blindness is quite rare. On the other hand, a temporary absolute blindness may occur as a part of uræmia without any retinal changes.

HÆMORRHAGES.

Partly from the change in the vessel walls, partly from the hypertrophy of the heart and the high arterial tension, hæmorrhages are rather frequent in Bright's disease. Retinal hæmorrhages have just been described, epistaxis is common, purpura and bleeding from the stomach and bowel may occur; the most important is cerebral hæmorrhage, which is a frequent cause of death in chronic renal disease.

SECONDARY INFLAMMATIONS, AND OTHER LESIONS.

Both in acute and chronic nephritis there is a tendency to inflammation of the serous membranes. Pleurisy is the most common; pericarditis is often the precursor of a fatal termination; acute peritonitis is perhaps more rare, unless it follows tapping the abdomen. Chronic inflammation of the peritoneum covering the liver (perihepatitis) has already been referred to in connection with Bright's disease. Bronchitis is a common, and endocarditis an occasional, complication; pneumonia not infrequently occurs towards the end. Finally, there are some lesions of the skin which may complicate Bright's disease—namely, (1) eczema; (2) an acute general dermatitis, with free desquamation, not unlike pityriasis rubra; (3) erythema læve, a condition of redness in more or less continuous patches, affecting the dropsical limbs; and (4) erysipelas.

URÆMIA.

This term is applied to a number of nervous symptoms that arise in different forms of Bright's disease.

Acute Symptoms.—The most striking of these are *uræmic convulsions*, or *uræmic eclampsia*. These have a very close resemblance to, indeed may be identical with, the ordinary attack of epilepsy. There is often a short tonic stage, and then general clonic convulsions of all the muscles of the limbs, face, eyes, and trunk. The face becomes livid, there is frothing at the mouth, and saliva may be tinged with blood, and the pupils are dilated. After some minutes the convulsions subside, and the patient lapses into a state of *coma*, from which he may again pass into convulsions; and these are repeated again and again with intervals of complete coma. During the convulsion the respiration is hurried, and the pulse is small and quick; the temperature is variable, and it may reach 104°, or higher. Thus, convulsion and coma succeed one another; but either may occur separately. Sometimes coma comes on quickly; or more slowly, drowsiness gradually increasing to stupor and complete unconsciousness in a few hours. A temporary *paralysis* is sometimes observed.

Delirium or mania is another form of disturbance which results from uræmia, but it is not so common as the convulsive and comatose symptoms; it may follow the fits.

It is also after convulsions that the blindness (*uræmic amaurosis*), to which I have already referred, generally occurs; it rarely precedes the fits, or happens without them. It may last from one to three days, and frequently passes off entirely. Deafness may be also noticed.

Chronic Symptoms.—These are headache, twitching of the muscles without loss of consciousness, recurrent attacks of *dyspnœa*, anxiety, and restlessness, or somnolence and stupor, itching of the skin, *vomiting* and *diarrhœa*. Vomiting may be referred to irritation of the nerve-centres, though it has been thought by some to be due to the presence of urea or carbonate of ammonium in the gastric secretion. The contents, however, of the stomach are usually acid. Diarrhœa, though often accompanying vomiting, is sometimes associated with decided lesions in the intestinal mucous membrane, which may be œdematous or ecchymosed, or in a condition resembling dysentery. The symptom can then scarcely be classed with the other uræmic conditions.

The dyspnœa sometimes resembles spasmodic asthma, and comes on at night. But I have twice seen cases in which stridulous breathing exactly resembling laryngeal obstruction or tracheal stenosis was only a part of uræmia, the patients being conscious at the time. And Fagge records a case which I take to be similar.

Results.—Though uræmic symptoms indicate a very grave condition, and generally coincide with a considerable diminution in the secretion of urine, and especially of urea, still they are often recovered from. This is especially the case in acute nephritis, where the condition of the kidney may be only temporary; whereas the less acute symptoms of uræmia in chronic nephritis not infrequently persist to the fatal termination. Here, also, an attack of coma often ends the scene; but it is generally a succession of fits rather than a single one that is fatal, whether in acute or chronic disease.

Pathology.—The most plausible explanation of uræmia is that it is due to the retention in the blood and tissues of some of the excrementitious matters that ought either themselves, or in some changed form, to be excreted by the kidneys; although it is quite unknown whether it is one substance alone, and if so, which of the many urinary compounds and derivatives. Analyses of the blood in uræmia have not generally shown a large proportion of urea; but it is found in the gastric and intestinal secretions, in the dropsical fluid under the skin, and it has been secreted by the sweat glands on to the surface of the skin, actually forming crystals (probably not entirely of urea), which give the skin the appearance of having been dusted with flour or pounded sugar. Such a case I have myself recorded ("Guy's Hosp. Rep.," 1874). But if urea be injected freely into the blood of animals it does not cause uræmia. Voit says, that if then water be withheld, so that the excretion of urea is hampered, uræmic symptoms appear. Landois produced convulsions by the direct application of kreatin, kreatinin, potassium phosphate, and some other salts to the surface of the brain in animals; but he obtained a negative result with urea.

Frerichs attributed the symptoms to an *ammonæmia*, from decomposition of the urea into carbonate of ammonium; but this has been disproved. Another suggestion is that the products of intestinal decomposition normally excreted by the kidney, may accumulate when that organ is diseased. The researches of Bouchard, among others, show that the several constituents of urine have different toxic effects, and that it is not always the same poison which causes the very various symptoms known as uræmic. Feltz and Ritter consider the potassium salts to be more poisonous than other constituents.

ACUTE NEPHRITIS.

Ætiology.—Exposure to cold is one of the most common causes of acute nephritis. It is more prejudicial when combined with damp, and is often immediately followed by the symptoms of nephritis, so that there can be no possible doubt of the association. Adults are much more liable than children to have nephritis from this cause, and the risk is increased if the individual is much exhausted from exertion or other cause, if he is asleep, or if he is under the influence of alcohol.

The poison of scarlatina is another exceedingly common cause of acute nephritis, and in this case children are more often the subjects than adults. As a rule, the symptoms are first observed during convalescence, and though some cases lend support to the idea that exposure to cold is the exciting agent, yet there can be no doubt that the nephritis arises in cases that have been most carefully nursed, and hence must be entirely the result of the preceding illness. Sometimes, indeed, it commences before convalescence is established.

Several other febrile diseases are from time to time, but much less frequently than scarlatina, the causes of acute nephritis. Of these cases also a very small proportion develop into a typical Bright's disease, with dropsy, secondary inflammations, uræmia, &c.; but the majority present only temporary changes in the urine, with but slight constitutional disturbance. They are measles, variola, cholera, varicella, typhus, enteric fever, relapsing fever, and erysipelas. Diphtheria is frequently accompanied by albuminuria, sometimes clearly indicative of nephritis. With these may be classed syphilis, and septic conditions from purulent absorption.

Pregnancy is not infrequently the cause of a nephritis, which may be of the most severe type, with uræmia and retinal changes.

A number of substances taken internally set up irritation of the kidney, which, according to the dose, may be severe congestion, or may amount to a definite nephritis. They are cantharides, turpentine, nitrate of potassium, salicylic and carbolic acids. Indulgence in alcohol, though probably more often contributing to the chronic form of nephritis, occasionally seems to produce an acute inflammation.

A more or less acute form of nephritis may occur secondarily to various lesions of the lower urinary passages (*consecutive nephritis*).

Symptoms.—These are in some cases almost confined to an alteration in the urine, of which the main feature is the presence of albumen; often, also, the urine is scanty, micturition is frequent, and blood and some casts may be present. With this there may be some febrile reaction, a feeling of malaise, and perhaps pain in the loins. In this latter case there can be no doubt of the existence of a nephritis, but if the urine is alone affected, it may be the result of severe congestion only, and it is important to remember that in the course of acute febrile illnesses a slight albuminuria occurs, which is probably due either to congestion or to a slight degenerative change in the renal structures.

But in the more typical cases, such as are commonly included under the name of acute Bright's disease, the first symptom is dropsy, which occurs in the manner described in the account of that symptom (p. 790). Sometimes it appears within a few hours after exposure to cold, or it is first noticed in the stage of convalescence from scarlet fever. Almost simultaneously, the urine becomes affected, and there may be a rigor and some discomfort, or pain in the loins. The urine is scanty, the quantity diminishing to ten, eight, or six ounces daily, or even less; it is acid and irritating, so that it is frequently voided in quite small quantity; its specific gravity is high, from 1025 to 1030; it is turbid, and it has a colour which is due to the presence of fresh or altered blood, and is dusky brown, deep brown, "porter-coloured," pink, or distinctly red, according to its quantity and condition. It deposits a sediment consisting of fresh or altered blood-corpuscles, or fragments of them, renal epithelial cells, hyaline, granular, epithelial, or blood casts, granular *débris*, and it may be after some time uric acid crystals. Albumen is always present, and generally in large quantities, forming a thick curdy deposit on boiling; or the urine may become actually solid on heating, so that the test-tube may be inverted without a drop running out. The amount of albumen in any specimen varies from 2 to 5 per cent., and the quantity discharged daily from 100 to 400 grains. It must be understood that this is, in most cases, far in excess of what would correspond to the small amount of blood contained in the urine. The urea is remarkably diminished, it may fall to half its normal quantity or less daily, and the phosphates and chlorides are also reduced.

Quite early the pulse becomes hard and tense, and the heart-sounds are modified as described. In severe cases the heart dilates, and the impulse may be displaced outwards.

In many instances this constitutes the whole of the illness. After a few days, or a week or two, improvement sets in, the dropsy begins to diminish, the blood disappears first from the urine, then after some longer time the albumen; the specific gravity diminishes, the quantity is increased, reaching, it may be, sixty, seventy, or eighty ounces daily, and eventually, with the exception of some pallor and weakness, the patient returns to his normal condition.

But the course of acute nephritis may be less favourable in several ways. In some cases quite early the urine is almost entirely suppressed. For several hours, or a day, not a drop is passed, or only a few drachms of dark brown, opaque, highly albuminous urine. Under these circumstances uræmic convulsions are very apt to occur, which may be fatal, or subside coincidently with an increase in the secretion of the urine.

In other cases dropsy becomes extreme, and effusion takes place into the peritoneum, pleural cavities, and pericardium. The lungs at the same time are partly œdematous, partly compressed by the pleural fluid, and death may take place from interference with their functions. Sometimes the excessive distension of the skin, especially in the lower extremities, causes gangrene and sloughing, or erysipelas occurs around the punctures which have been made to relieve tension; and death in either case may follow.

In other instances, again, the acute serous inflammations, pleurisy, pericarditis, and peritonitis, are fatal; or acute œdema of the glottis brings about asphyxia, unless promptly relieved by tracheotomy. But there is scarcely any of these serious dangers from which recovery may not take place, and sometimes, after living for months on the verge of death, the patient may ultimately get quite well. Lastly, the condition may remain for a long time unchanged, and eventually it becomes subchronic or chronic, and must be classed with the form of nephritis to be described presently.

Morbid Anatomy.—In some cases, especially those in which the glomeruli are mainly affected, the kidney may be but little larger than normal; the cortex is dark, and the glomeruli are pale. In other cases the kidney is more or less swollen, it may be twice its normal size, it has a rounded form, is tense and elastic, the capsule strips readily, and the surface is somewhat paler than normal. On section, the cortex is considerably swollen, and of greyish-red colour, while the pyramids are dark red from congestion. Here and there are bright red spots: some of them are congested glomeruli, others are small hæmorrhages. In yet other cases the kidneys are intensely congested, dark red or almost chocolate in colour, dripping with blood, and showing on section a still more extreme congestion of the pyramids.

Microscopically one can distinguish changes in the tubules, in the interstitial tissue, and in the glomeruli, which may all be present together; and if one speaks of a glomerular, of an acute interstitial, or of a tubal nephritis, it must be understood that, though one of these structures is especially concerned, the others are probably affected, though in a very much less degree.

In the tubules the epithelial cells become swollen and granular, and in more advanced conditions are filled with fat granules, or become necrosed, and are separated from the wall of the tubule, (desquamation). The tubule is then filled or even distended by accumulations of such altered cells, mixed with albuminous fluid, leucocytes, and granular *débris*, and here and there blood escapes into them from distended intertubular vessels. Many tubes, both in the cortex and in the pyramids (*e.g.*, Henle's loops), are filled with hyaline or other casts. The distension of the tubes gives a pale colour to the portion of the kidney affected, partly from the opacity of the fat granules, partly from compression of the intertubular vessels; on the other hand, such compression increases the congestion of the glomerular tufts.

The size of the largest kidneys is perhaps due to interstitial exudation. This may be either a general infiltration with inflammatory fluid, or the extravasation, more or less irregularly, of leucocytes from the blood-vessels. They are most abundant around the Malpighian capsules, and the intertubular vessels. The glomerular changes (*glomerular nephritis*) consist of the distension of the Malpighian capsule with a fine granular mass in which are embedded numerous small angular nuclei. Occasionally, hæmorrhage takes place. The glomerular tuft is compressed by the effusion, and the circulation and secretion of water are materially interfered with. A hyaline transformation of the walls of the afferent arteries has also been described as a constant change in the glomerular nephritis of scarlatina.

Diagnosis.—This rarely presents any difficulty. The sudden appearance of a general dropsy, with scanty, albuminous urine, in one previously quite healthy, or recovering from scarlatina or other fever, can scarcely be mistaken for anything else. In the absence of dropsy, the mere presence of albumen in the urine is not sufficient for the diagnosis. Turbid brown urine, abundance of albumen and casts, and constitutional disturbance will then show that there is nephritis, but for this the name of Bright's disease would not be generally employed. If there is a small quantity of albumen only in the course of an acute disease, this may be due to a change in the epithelium, not usually regarded as constituting nephritis, though it is not essentially different from what actually takes place in the typical conditions. Lesions of the *lower urinary passages* may cause bloody urine and albuminuria. Dropsy and casts are absent. *Hæmoglobinuria* causes a red urine, in which, however, there are no blood-corpuscles. Rarely the dropsy of nephritis occurs without albuminuria, especially in children. Such cases must be distinguished from some insidious diseases (tuberculosis, leuchæmia), which have other characteristic lesions.

Prognosis.—Indications of this have been already given. It is, on the whole, favourable, as there are a great number of mild cases which recover in a few weeks, and even after months of severe symptoms complete cure may result. Other cases are fatal in the acute stages, or run on into chronic nephritis. The most frequent causes of death are hydrothorax, uræmia, and inflammations such as pneumonia, pericarditis, and, more rarely, peritonitis.

Treatment.—The direct treatment of the inflamed kidneys, either locally or by drugs, is, as a rule, not attempted. Only exceptionally, where there is much lumbar pain or free hæmorrhage from the kidneys, may dry-cupping be employed to the loins, or for the former symptoms a hot poultice or fomentations. The treatment of nephritis consists in keeping the patient at rest, removing all sources of irritation, limiting the supply of food likely to put a strain upon the excreting functions of the kidney, and supplementing these functions as far as possible by the use of other secreting organs—the skin and the bowels. In addition, we have to deal with special symptoms and complications as they arise. Rest in bed in a warm room diminishes the strain upon the heart and the need for highly albuminous food, while the secretion from the skin is likely to be promoted.

The diet is best confined to milk alone, or milk and soda-water. All kinds of meat, fish, and eggs should be rigorously excluded, and only in milder forms or after the acute stage has passed may a moderate amount of farinaceous food—gruel, arrowroot, and, later, toast, and a little bread and butter—be allowed, and with progressing convalescence a gradual return to meat diet, through beef-tea, broths, eggs, and fish.

In mild cases the medicinal treatment should be a saline diaphoretic, such as liquor ammonii acetatis or liquor ammonii citratis, in doses of 3 or 4 drachms every four hours; and the bowels may be kept active by occasional purges of pulvis jalapæ co., 20 to 40 grains, or senna and sulphate of magnesia. Lemonade may be given as a beverage, or the Imperial drink, which may act mildly on both the bowels and the skin. This consists of a drachm or a drachm and a half of cream of tartar dissolved in a pint of boiling water, flavoured with lemon peel and sugar, and drunk, *ad libitum*, when cold.

If dropsy is considerable, or if there are fears, either from the scantiness of the urine, or from headache, drowsiness, and twitching, that uræmic convulsions are imminent, the treatment by purgation and diaphoresis must be more decided. Diaphoresis should be promoted by the vapour bath, by the hot-air bath, by the use of jaborandi, or by pilocarpine. The *vapour bath* is administered by raising the bed-clothes from the patient by means of a low cradle, and fitting them close about his neck and round the sides and end of the bed. Into the space thus formed projects the long tube from a kettle of water, placed at the foot of the bed, and kept boiling by a spirit-lamp or gas-flame. The steam thus brought into contact with the patient's body promotes free perspiration. Or a *hot-air bath* may be given by burning a spirit lamp under a funnel connected with a tube similarly placed. The exposure should be from fifteen to twenty minutes; but it is desirable to take the temperature of the patient, as it may be inconveniently raised, if free sweating does not occur.

Jaborandi may be given in doses of 1 or 2 drachms every four hours; but the most efficient diaphoretic is the nitrate of pilocarpine, of which $\frac{1}{6}$, $\frac{1}{4}$, or $\frac{1}{3}$ grain should be injected subcutaneously once a day.

In extreme dropsy, especially where the skin is tense and threatens to become inflamed or to slough, the dropsical fluid may be removed either by small incisions with a lancet or punctures with the needle, or by the use of Southey's tubes. Two or more may be placed in each leg, and by this means several pints of serum may be withdrawn in a few hours. Only occasionally, in extreme cases, is it desirable to tap the abdomen or to aspirate the pleural cavity.

If uræmic convulsions set in, the patient's tongue should be protected, as in epilepsy, by placing a piece of stick between the teeth, and some pilocarpine should be at once injected under the skin. A few whiffs of chloroform will check the convulsions quickly, and though they may return on the removal of the anæsthetic, the effect may be kept up by its occasional use in small quantities, and thus a great deal of the violence of the fit is prevented. Bleeding from the arm will also sometimes stop them, and though it is, as a rule, undesirable that patients with pronounced renal disease should lose much blood, this remedy may be properly used if the convulsions are violent and persistent. The blindness that may occur after convulsions generally recovers of itself. Coma should be treated by purgation and the use of pilocarpine. Vomiting will require effervescing mixtures, dilute hydrocyanic acid, or tincture of iodine in 3 or 5 minim doses every hour.

During convalescence iron should be given to restore the condition of the blood, but it is not well to use it in the early stages. The perchloride, and the ammonio-citrate, are suitable preparations. Persistent albuminuria has been treated by various astringents; but rarely with much success. Saundby states that he found albumen most decidedly lessened by Imperial drink, citrate of lithium, bicarbonate of potassium, and the bicarbonate, benzoate, or tannate of sodium. Lactate of strontian (gr. 20 to 30 ter die) has recently been tried. The patient throughout should be carefully kept from cold, and should, on getting up, wear warm clothing, with flannel next the skin.

CHRONIC TUBAL NEPHRITIS.

This includes two stages, in one of which, the large white kidney, the change is really most marked in the renal epithelium; while the second, or the pale granular kidney, is due to interstitial changes, which are progressing, *pari passu*, with those in the tubes.

Ætiology.—This condition is by many described as exclusively a late stage of acute nephritis; but since it sometimes occurs spontaneously, and often insidiously, it seems better to treat of it independently. In cases that are chronic from the first the ætiology is by no means always clear. It has been attributed to cold and damp acting over long periods of time, as, for instance, from residence on damp soils; and a predisposition to its occurrence possibly arises from habits of intemperance, overwork, mental exertion, or sexual indulgence. Toxic and infectious agencies have been suggested as causes, but it is not evident what these are in particular cases; and if malaria, syphilis, and tuberculosis sometimes seem to be exciting agents, it may be remembered that they are also related to the lardaceous disease of the kidney, of which we shall have to speak presently. Cases of this kind are more common in persons of middle age; rare in children and old people; and males are more liable than females.

Symptoms.—If the disease follows acute Bright's disease, there is a continuance of the symptoms already described:—General dropsy, effusion into the serous cavities, scanty albuminous urine, high tension of the pulse, and tendency to cardiac hypertrophy or dilatation. Or there may be an apparent recovery from the acute attack, and after a short interval the symptoms recur.

In primarily chronic cases the beginning is more or less insidious. Pallor, loss of appetite, nausea, headache and frequent micturition are the first signs, and then oedema may be observed in the lower extremities at night, and around the eyelids in the morning. The dropsy gradually increases, and the case is then almost identical with the acute disease.

The urine is scanty, highly albuminous, sometimes smoky, is deficient in urea, and contains casts. Its quantity varies from ten to twenty-five ounces, though in later stages for a time it may be more abundant. The albumen forms a precipitate which occupies one-third to one-half of the urine boiled, really amounting to 2 or [illegible] per cent. by weight, or a daily discharge of from [illegible] to [illegible] grains. The specific gravity is at first rather high ([illegible] to 1025), but later, with the progress of the disease, it becomes much lower. The casts are granular, hyaline, and epithelial, and fatty casts are numerous in the cases of large white kidney with much fatty degeneration of the renal epithelium. They are accompanied by leucocytes, renal epithelium, pus, and granular *débris*. In some cases the urine is frequently bloody (*chronic hæmorrhagic nephritis*). The amount of urea is always below the normal, and has the same importance with reference to the onset of uræmia as in the acuter forms of disease.

As the case progresses the heart becomes hypertrophied, and the vessels are thickened. The impulse of the heart is displaced outwards, but the oedema of the thorax may prevent its being felt; the aortic second sound is accentuated, the first sound is frequently reduplicated, and the pulse is one of high tension. The retinal changes already described are observed in a certain proportion of cases, while secondary inflammations, chronic uræmia, or uræmic convulsions and coma may at any time occur.

If the patient is not carried off in the earlier stage, the kidney becomes contracted, and some alteration in the urine takes place—that is, a relatively larger quantity of water is secreted, and the urine has a lower specific gravity. Ultimately, cardiac symptoms may become the prominent feature of the illness, as in chronic interstitial nephritis.

The course, in the majority of instances, is to a fatal end, brought about in the same way as in acute nephritis, by uræmia, pleurisy, pericarditis, pneumonia, œdema of the lungs, œdema of the glottis, increasing dropsy, or inflammation or sloughing of the integuments; and this is commonly in from six to eighteen months, though some cases go on for two or three years. A few cases even after this period of time may appear to recover completely.

Morbid Anatomy.—Chronic tubal nephritis is seen in two forms or stages—the *large white kidney*, and the *pale granular kidney*. *Chronic hæmorrhagic nephritis* is a variety of the former.

The *large white kidney*, or inflammatory fatty kidney, is larger than normal—the two together may weigh as much as twenty-eight ounces (Fagge), often they [illegible] sixteen. It is smooth on the surface, and the capsule strips easily; it is of yellowish or greyish-white colour, and covered with venules radiating from a central point (stellate veins). On section, the cortex is broader than normal, of the same colour as the surface with an appearance of coarse striation, while the pyramids are more or less dark-red. Here and there are red spots, due to hæmorrhage into the tubes, and these are in some cases so abundant as to justify the term *hæmorrhagic nephritis*. Microscopically it is seen that the convoluted tubes are filled with epithelium in a state of fatty and granular degeneration, desquamated from the surface, as well as separate fatty granules; epithelial and granular casts occupy the straight tubes. This distension of the tubes with opaque material, and the compression they exert on the vessels, account for the white or grey colour of the cortex. The intertubular tissue is irregularly infiltrated with leucocytes, and the Malpighian capsules are thickened. Lastly, within the capsules, the glomerular tufts are compressed by exudation and the proliferation of the epithelium covering them.

In the later stage, that of the *pale granular kidney*, or secondary contracted kidney, the organ is about the normal size, or not much smaller. Its surface is rough or "granular," the capsule slightly adherent, the colour yellowish white, or more or less mottled with red in the areas of depression between the granules. The cortex, on section, is less broad than in the earlier stage, may become somewhat narrower than normal, and is not so white as in the "large" kidney. Microscopically, the intertubular tissue is partly infiltrated with leucocytes, partly changed into connective tissue; and it is by the contraction of this tissue that the granular condition is brought about, and the organ becomes smaller. Of the tubes some still contain altered epithelium and *débris*, others are atrophied from pressure, from others again small cysts have been formed, similar to those so common in the red granular kidney. Many glomeruli, also, are atrophied, or changed into fibrous nodules; and the small arteries are thickened. Lardaceous changes of slight extent are sometimes present.

Diagnosis.—The distinction between an acute nephritis and the first stage—large white kidney—of the chronic nephritis, which succeeds it, must be purely arbitrary; from four to six months may be regarded as the duration of the acute condition. In any case of less duration, the diagnosis must depend on the history of sudden or insidious onset, the knowledge of a cause, such as scarlatina, or its absence.

After a longer period, chronic tubal nephritis may be confounded with chronic interstitial nephritis, with lardaceous disease, or with the albuminuria of primary heart-disease (*cyanotic induration*). From *chronic interstitial nephritis* it is distinguished by its history, the early appearance of dropsy, the age of the patient, which is mostly younger, the early scantiness of urine with abundant and often fatty casts; and from *lardaceous disease* by the absence of such causes as prolonged suppuration, phthisis, or well-marked syphilis, and by early scantiness of urine and abundant deposit. The similarity between this and heart-disease may be close, though not so much so as between heart-disease and chronic interstitial nephritis. This subject will be discussed more fully under the last head. Here it may be sufficient to state that the history, the general character of the dropsy, and the mostly large quantity of albumen will help to distinguish primary renal disease from secondary albuminuria.

Treatment.—This is not essentially different from that of acute nephritis. Rest in bed, flannel clothing to promote warmth and a free action of the skin, and milk diet, are absolutely essential in the early stages. The bowels must be kept active, and diaphoresis excited. The treatment for urgent symptoms is the same as for acute nephritis, except that venesection may have to be used with more caution in view of the marked anæmia. This last symptom in prolonged cases should be met by the use of iron preparations, such as the iodide, tartrate, or ammonio-citrate. In the more chronic forms, with albuminuria but little dropsy, benefit may be derived from residence in warmer climates—Bournemouth or Tenby in the British Isles, the south of France, Italy, or Egypt.

CHRONIC INTERSTITIAL NEPHRITIS.

(*Cirrhosis of the Kidney, Red Granular Kidney, Primary Contracted Kidney, Gouty Kidney, Indurative Nephritis, Renal Sclerosis.*)

Ætiology.—Chronic interstitial nephritis is a disease of middle and advanced life; it occurs with extreme rarity under twenty years of age, and most frequently between forty and sixty. It affects males more than females. From its very slow development, it might be expected that the causes would be such as were in continuous action. The most important are gout, whether latent or developed, and chronic lead-poisoning. Alcohol, also, has considerable influence; sometimes, perhaps, by inducing the gouty habit. Some cases may be due to heredity, to chronic dyspepsia, or to climatic influences. Dickinson points out that renal diseases are more common in temperate climates, with their greater atmospheric variations, though these seem more likely to produce acute nephritis. There remains a good number of cases in which the cause cannot be clearly made out. Obstruction to the passage of urine in the ureter, bladder, or urethra, is followed by a chronic interstitial inflammation of the kidney (*consecutive nephritis*).

Anatomy.—The kidneys are reduced in size, sometimes to a remarkable extent, and often one more so than the other; the two organs together may only weigh three or four ounces, and Fagge gives, as an extreme instance, a pair recorded by Wilks as weighing thirty grains under one ounce and a half. The shape of the kidney is not materially altered, except from some irregularity in the rate of contraction in different parts. The capsule is adherent, and stripped off carries with it small portions of renal tissue; the whole surface of the kidney is then seen to be covered with minute elevations (granulations) of $\frac{1}{16}$ to $\frac{1}{8}$ inch in size, with intervening depressions, and here and there may be cysts varying from $\frac{1}{8}$ to $\frac{1}{3}$ inch in diameter, and containing a clear fluid or colloid material. The colour is mostly brownish-red, or dark red or pink, varying somewhat with the vascularity and the amount of interstitial tissue. The whole organ is tough; on section the cortex is found to be very narrow, sometimes reduced to $\frac{1}{4}$ or $\frac{1}{8}$ inch; the pyramids are also smaller than normal, and generally somewhat darker in colour than the cortex. Cysts may be present in the substance of the organ, and the divided vessels often stand out prominently on account of their abnormal thickness.

On microscopical examination it is seen that the anatomy of the organ is much altered. Here and there are patches infiltrated with leucocytes; these are most abundant in recent cases. In very old cases they have given place to the fibrous tissue, by the contraction of which the kidney has been so reduced in size. The Malpighian capsules are thickened; many of the tufts are atrophied, and converted into fibrous nodules; and from the atrophy of intervening tissue the glomeruli are often crowded together irregularly.

In earlier stages there is some degeneration of the epithelium of the tubules, similar to that of the acuter forms, but slower in its course; and shed epithelium, leucocytes, and hyaline casts are seen in different tubes. Later the tubes become atrophied, partly by pressure of the new connective tissue, partly in consequence of the atrophy of the glomeruli, whose absence must deprive the tubes of their function, both by the non-secretion of water, and by the obstruction to the intertubular circulation which the atrophy of a glomerulus carries with it. The cysts arise from local obstructions to the tubes.

In the above description it is assumed that the lesion is chiefly and primarily inflammatory, though of very slow development; but it is right to mention that such a view is not held by all. Strümpell regards the lesion as essentially degenerative, beginning in the epithelium of the tubules and the glomeruli, and followed by secondary infiltration and contraction. Greenfield believes that the first change takes place in the glomeruli, and that their atrophy necessitates the other changes. Ziegler and other German writers speak of an *arterio-sclerotic kidney* as different from the kidney of chronic interstitial nephritis. The first change takes place in the arteries, which become thickened, obstruct the blood-supply, and hence lead to atrophy. There is, according to them, no induration. It occurs in old people, and in chronic lead-poisoning. This form is clearly included by English writers under cirrhosis and granular kidney.

Symptoms.—The onset of chronic interstitial nephritis is generally quite slow, and marked by few distinctive features. Often, indeed, the kidneys are found granular in patients who die of other diseases, or one is struck down by cerebral hæmorrhage, without any symptom having attracted attention to the condition of these organs. Amongst early symptoms, which, occurring in a middle-aged person, should make one think of granular kidney, if not accounted for in other ways, are recurring or persistent headache, nausea and vomiting, shortness of breath, anæmia, loss of appetite, and general weakness. Often quite early it may be elicited, that the patient is passing a large quantity of water, especially that he has to get up frequently at night to empty his bladder, and that the urine is pale and abundant. Occasionally no symptom may be sufficiently prominent until the sight is affected by renal retinitis, and the patient's eyes are examined with the ophthalmoscope.

The urine is quite normal at first; but in course of time it becomes abundant, pale in colour, almost watery, and of low specific gravity, 1005 to 1012. The solids, including the urea, are below the normal. The quantity of albumen is small, often a mere trace, generally not more than ·5 per cent. It may be for days entirely absent, or it is more abundant at one time in the day than another. The urine is quite clear, or throws down a very scanty deposit, in which a few hyaline or granular casts may be found. There is not at first any dropsy. Even after albuminuria has been recognised some time, there may be no more than a slight œdema of the ankles, or a little puffiness of the eyelids, or a watery conjunctiva. If the dropsy becomes considerable, it either has the characters of the cardiac form, and is the result of the secondary implication of the heart, or it is a renal dropsy, and is due to the supervention of acute nephritis upon the chronic disease. The heart and pulse reveal the changes in the circulation that have been described. The impulse of the heart is displaced outwards, the aortic second sound is accentuated, and the first sound is perhaps reduplicated. The pulse is hard and cord-like; the arteries may be rigid and atheromatous.

In such a condition the patient may continue for several months or years, with varying health, but without any serious symptoms. But finally life is endangered by the several complications which have been indicated in the summary at the beginning of this chapter. *Cerebral hæmorrhage* carries off a certain proportion of cases. It may occur in the cerebrum, in the pons, or in the meninges, and results in apoplectic coma or paralysis of varying degrees, according to its extent and situation. *Pleurisy*, *pericarditis*, and *pneumonia* not infrequently occur, and more rarely peritonitis. Uncontrollable *vomiting* is often the cause of death, and sometimes constant *diarrhœa*. The symptoms called chronic uræmia are more frequent than coma or convulsions.

The most important feature of chronic Bright's disease, from the clinical point of view, is its effect upon the *heart*. This organ at first hypertrophies, to overcome the resistance in the arterioles, but subsequently dilatation is added. As this becomes more pronounced a systolic murmur is produced at the apex either from eddies set up in the dilated ventricle, or from actual regurgitation through a widened mitral orifice, for which the valve has become insufficient. Finally, the heart becomes irregular in its action, the other cavities are involved in turn, and a condition of in efficiency is produced identical with that of mitral valvular disease. Pulmonary congestion, perhaps with hæmorrhage, hepatic engorgement, ascites, and dropsy of the lower extremities follow upon this, and the urine is altered in the same manner as it is by a primary valvular disease—that is to say, it becomes scanty, high-coloured, and deposits urates, thus entirely losing the characteristics of the urine of granular kidney. The pulse may for some time retain its hardness, but eventually in some cases may become indistinguishable from the pulse of mitral disease. Finally, death may take place from cardiac failure.

Diagnosis.—Chronic interstitial nephritis must not too hastily be assumed from the detection of a small quantity of albumen in the urine of a middle-aged person. The early symptoms must be carefully inquired into, the heart and pulse investigated, and the urine examined on several occasions. In their typical conditions there can be no difficulty in distinguishing chronic *tubal* nephritis from cirrhosis of the kidney. In the former there are general dropsy, scanty urine, much albumen, and casts of all kinds; in the latter we see little or no dropsy, much urine, a small quantity of albumen, few or no casts, and there may be a very long history. But in the later stages of tubal nephritis, it may more closely resemble cirrhosis, and cirrhosis may be complicated by an acute tubal attack. The history will be of much importance in each case. It is often very difficult, or even impossible, to determine whether a case is one of *primary heart-disease* with albuminuria from congested kidneys, or one of cirrhosis of the kidney with secondary dilatation and hypertrophy of the heart. One must look for data in the history, the condition of the urine, the pulse, and the nature of any cardiac murmurs. It has already been said that the urine of chronic renal disease may in time assume all the characters of heart-disease; nor can the pulse be absolutely relied upon. A localised systolic murmur at the apex may occur in both cases; diastolic or presystolic mitral, and much more aortic murmurs, would be in favour of primary heart-disease. A mitral regurgitant murmur heard behind would be of doubtful significance. Previous attacks of rheumatism or of cardiac trouble from which the patient had temporarily recovered would be somewhat in favour of valvular lesions.

Prognosis.—This is unfavourable, but much improvement may take place, and life may be prolonged in some cases to an advanced age. The greater the extent to which the heart is implicated the shorter is the expectation of life, and the occurrence of retinal changes is generally the indication of a short future.

Treatment.—After the removal of any condition which can be safely regarded as the cause of the nephritis—*i.e.*, alcohol, lead, constant exposure to cold, &c.—the objects should be (1) to diminish the call upon the excretory power of the kidney; (2) to reduce arterial tension, and the consequent strain upon the heart and vessels; (3) to remove anæmia; and (4) to treat special complications as they arise.

More or less perfect rest, milk diet, the use of diaphoretics, and occasional purgatives will fulfil the first indication. Not that absolute rest is practicable throughout the whole course, which may be many years, but it may be enforced when symptoms are serious, and at all times over-exertion and strain should be avoided. Similarly, at times of comparative health, or improvement, the diet may largely consist of milk and farinaceous food. Meat should be reduced to a minimum, and alcohol should be entirely stopped, or amount at most to a glass of claret or dry sherry. The patient should be warmly clothed, and residence in a genial climate is of great benefit.

The second indication is also fulfilled in the same way; but it may also be assisted by the use of nitro-glycerine, given in doses of $\frac{1}{100}$ minim daily, or twice daily, and gradually increased. This drug reduces tension by dilating the arterioles, and often relieves the troublesome symptoms of headache and dyspnœa, which may occur in renal cirrhosis. Purgatives and diaphoretics both reduce tension, and assist in eliminating materials that are normally excreted by the kidneys.

Various preparations of iron should be given for the anæmia.

Among the complications which require treatment are uræmia, headache, vomiting, and cardiac dilatation and failure. The treatment of uræmia has been already given (p. 802). For headache Ralfe recommends 20 drops of dilute nitric acid in water;

pilocarpine injections may also be employed. For vomiting, effervescing mixtures, dilute hydrocyanic acid, a few drops of tincture of iodine in water every hour, or a cold compress or blister to the epigastrium may be tried. For the cardiac symptoms, which result from dilatation and feeble contraction of the ventricle, digitalis should be given, the bowels should be kept open, and generally the case should be treated like one of heart-disease.

CONSECUTIVE NEPHRITIS.

It is well to deal here with a group of cases of nephritis already alluded to once or twice under the above title. They result, first from various diseases of the ureter, bladder, and urethra, by which the passage of urine is interfered with; for instance, calculus, the pressure of tumours on the ureters, tubercular growth in the ureter, hypertrophy and thickening of the bladder obstructing the orifices of the ureters, cystitis, and prostatic enlargement; secondly, from septic conditions of the same parts following upon stricture, cystitis, &c.

The following three forms may be recognised: they may, however, be associated together in the same kidney:—

Chronic Interstitial Nephritis.

This is due to the increased pressure of urine behind an obstruction. If the obstruction is of long duration, the ureter and pelvis of the kidney become enormously dilated, and a hydronephrosis is produced, with more or less atrophy of the renal substance; but in the earlier stages the kidney becomes indurated from chronic interstitial change. This stage is not infrequently seen in women after death from malignant disease of the uterus and vagina, which obstructs the ureter, but causes fatal symptoms too soon to allow the formation of a complete hydronephrosis. The kidneys are pale pink, or pale yellow, of about normal size, and very hard; on section, there is some dilatation of the pelvis and the calices, and a little wasting of the pyramids, while the cortex is relatively broad. Under the microscope there is found infiltration of the organ with leucocytes, chiefly around the Malpighian capsules, and in the intertubular tissue; there is also some glomerular change, with a slight alteration of the tubal epithelium. In certain cases the process may go on to contraction of the new tissue, and the production of cicatrices.

The **Symptoms** are not prominent, and the condition of the urine is liable to be masked by the primary lesions of the ureter and bladder. Albuminuria may or may not be present; the urine is generally abundant and of low specific gravity. The disease affects one or both kidneys, according to the position of the obstructing lesion.

ACUTE OR SUBACUTE DIFFUSE NEPHRITIS.

This is mainly an acute interstitial and glomerular nephritis, associated in some cases with inflammation of the pelvis of the kidney, or pyelitis (M. Beck). Both kidneys are affected. They are swollen, pale yellow, or yellow mottled with red, with prominent stellate veins. On section, the cortex is pale, or mottled, the pyramids often dark. Under the microscope, the intertubular tissue and the space between the Malpighian capsule and the tuft of vessels are seen to be crowded with round cells, but the tubules show comparatively little change, only some granular epithelium, with round cells which may have extravasated from the interstitial spaces.

This form of nephritis is sometimes set up by operations about the urinary organs, or by washing out the bladder; in which cases the preceding condition of high pressure or of sepsis will obviously act as a predisposing cause.

The **Symptoms** begin suddenly with chill and rigor; and often there is moderate fever of intermittent type, the temperature being normal in the morning, and from 99° to 101° in the evening. The urine is natural in amount, or abundant if there has been increased urinary pressure for some time previously. There is a small quantity of albumen, and there may be hyaline casts; but often these data are masked by the condition of the lower urinary passages—*e.g.*, the presence of cystitis. The general symptoms are weakness, languor, drowsiness, thirst, loss of appetite, nausea, and occasional vomiting. Improvement may take place slowly, or death from more acute nephritis, from suppurative nephritis, from exhaustion, or some intercurrent disease.

SUPPURATIVE NEPHRITIS.

When suppuration of the substance of the kidney is secondary to lesions of the lower urinary passages, it is frequently associated with pyelitis, and hence is often termed *pyelo-nephritis*. All those causes which produce obstruction to the passage of urine, distension of the urinary passages, and cystitis, may ultimately result in suppurative nephritis; for instance, stricture of the urethra, prostatic enlargement, and calculus; various diseases of the pelvic organs, uterus, ovaries, and appendages in the female, which involve the bladder or ureters; and paralysis of the bladder from spinal injury, spinal disease, or myelitis.

Anatomy.—The kidney is usually enlarged and softened. The surface is mottled pale yellow and red, and presents several small points of suppuration, some of which may be torn open when the capsule is stripped off. On section, the cortex shows the same mottled colour, the pyramids are usually red and congested, and the kidney has the appearance of one in a state of acute interstitial nephritis. The characteristic feature is the presence of numerous yellow streaks of pus, stretching in a radial direction from the surface inwards through the cortex, and even into the pyramids. They are sometimes wedge-shaped, or conical, with the base at the surface, in other cases simply linear. Under the microscope the kidney is seen to be acutely inflamed; there are numerous leucocytes in the intertubular tissue, some glomerular inflammation, and swelling and nuclear proliferation of the epithelium of the tubes.

In the majority of cases, no doubt, infection by micro-organisms takes place upwards along the ureter, pelvis of the kidney, and renal tubules. In other instances the infection is conveyed by the blood-vessels or lymphatics.

Symptoms.—It will be understood that the patient who suffers from this form of suppurative nephritis is generally already the subject of stricture of the urethra, enlarged prostate, cancer of the uterus, or of cystitis from retention of urine in spinal disease. The first symptoms are often chills or rigors, with a rise of temperature to 104°, 105°, or 106°, and there may be a regular intermittent or remittent fever. Then follow the characteristics of a typhoid or septicæmic condition—loss of appetite, dry brown fissured tongue, nausea and vomiting, sometimes diarrhœa, sweating, and rapid emaciation. The patient is somnolent, in a more or less dreamy state, but without actual coma or delirium, till quite the last. The pupils vary, and have no special significance. The pulse is quick and feeble. Lumbar pain is sometimes present, and there is generally some tenderness on deep pressure in the loins. The state of the urine is determined by the preceding disease; it may thus contain pus, mucus, blood, or albumen; and it may be difficult to recognise any additional albumen, or even the hyaline casts, or the epithelial cells which result from an acute nephritis. In some cases, on the other hand, renal and pelvic epithelium and hyaline casts are found in sufficient quantity. The urine is often abundant, and the urea not much diminished; a considerable decrease may be due to preceding chronic nephritis. H. Morris notes that in cases with intermitting fever, the quantity of urine is often greater during the febrile periods than between them.

The duration of the illness is not more than four weeks, and may be so short as three or four days.

Diagnosis.—The occurrence of pyelo-nephritis in cases of disease of the bladder and ureters is generally signalised by the fever and the typhoid condition. It is likely to be confounded with pyæmia, other forms of septicæmia, enteric fever, peritonitis, and ague. In *pyæmia* one looks for secondary abscesses, which are absent in pyelo-nephritis. The temperature of pyæmia shows a more extensive range, and the rigors are followed by profuse sweating. Milder forms of *septicæmia* are almost indistinguishable from pyelo-nephritis; in the acute forms there are more restlessness and anxiety. Purpura and internal ecchymoses do not occur in pyelo-nephritis. In *enteric fever* the typhoid state is more slowly developed. Rose spots, typical motions, and the regular curve of temperature are in its favour; while rigors and an irregular fever would speak strongly for suppurating kidney. *Peritonitis*, which may arise from lesions of the bladder, is recognised by the absence of rigor, the greater pain, and more severe vomiting. In *ague*, the intervals between the rigors are apyretic and almost healthy.

Treatment.—That suppurative nephritis may supervene, should always be borne in mind in the treatment of diseases of the urinary organs; and it should, as far as possible, be prevented by dealing with its cause. Where cystitis is the main difficulty, as in spinal cases, the bladder should be regularly emptied by the catheter, and irrigated with some antiseptic solution, such as salicylate of sodium (5 gr. to 1 oz.), quinine, (1 or 2 gr. to 1 oz.), or borax (5 gr. to 1 oz.). When suppurative nephritis has declared itself, treatment is of little avail. The patient should be supported by nutritious but easily-digestible food. The action of the skin should be promoted by vapour or hot-air baths. If there is much lumbar pain, hot sand may be placed on the loins, or dry-cupping may be employed. The bowels should be kept active. Internally, quinine, with small doses of opium or liq. morphinæ, seems the best remedy to give.

METASTATIC NEPHRITIS.

Metastatic abscesses occur in *pyæmia* and occasionally in *malignant endocarditis* as the result of micro-organisms being conveyed by the blood-vessels to the kidneys.

Pyæmic abscesses are generally scattered through the cortex of the kidney; are of small size, more or less elongated, and sometimes conical; and are surrounded with a red zone of vascularity. Their occurrence does not obviously add to the symptoms of pyæmia. Albuminuria may occur independently of them, and cannot therefore be held to indicate their presence.

In malignant endocarditis, the abscesses may arise from the breaking down of embolic infarcts of various sizes. These are conical, but generally have a different shape from the abscesses of pyelo-nephritis, the base being relatively broader. Embolic infarction may be indicated by pain and by blood and albumen in the urine, but albumen occurring alone in the course of endocarditis does not of necessity indicate suppuration.

Sometimes, both in pyæmia and endocarditis, there may be one or two large abscesses, instead of several small ones.

PERINEPHRITIS AND PERINEPHRIC ABSCESS.

Perinephritis is the term used for inflammation of the cellular and adipose tissues around the kidney.

Ætiology.—It arises:—1. From injury—such as blows, kicks, or strains. 2. From the extension of inflammation from the kidney, the pelvis of the kidney, or the ureter. This may be the result of suppurative pyelitis, pyonephrosis, tubercle of the kidney, or calculus, which either sets up pyelitis, or itself ulcerates through the kidney or pelvis. 3. From inflammation, especially suppuration, in more distant parts spreading to the perinephric tissue; for instance, pelvic cellulitis, perityphlitis, abscess of the liver or spleen, caries of the spine and psoas abscess, or inflammation of the gall-bladder.

Pathology.—The perinephral tissue in different cases undergoes all the changes that may take place in other inflamed tissues. In early stages it is vascular, œdematous, and infiltrated; then points of suppuration occur, and finally one large abscess is formed. The pus is free from odour, or very offensive; or it has a fæcal odour, from contiguity with the bowel. Sometimes shreds of gangrenous tissue are present. The kidney may be infiltrated or softened in the middle of the abscess. Occasionally the perinephral tissue is indurated by a more chronic process.

Symptoms.—These are to a certain extent those which accompany other inflammatory processes. The onset may be insidious, when there is nothing but some dull aching *pain;* in other cases it will be marked by rigor, with elevation of temperature, which continues uniformly high, or is intermittent in character. The pain is deep-seated, in the loin or side of the abdomen, and radiates to the hypogastrium, groin, or genitals. The pain in the loin is increased by pressure, and on examination a certain amount of fulness or resistance may be felt in that region. This examination should always be conducted by the bimanual method described in the examination of the abdomen (p. 583).

As the case progresses, a more or less extensive *tumour* occupies the space between the last rib and the crest of the ilium, uniformly dull, bulging the flank, causing œdema of the loin, and perhaps fluctuating. The leg of the same side is often flexed at the hip-joint, and attempted extension causes pain; attention has also been called (by Gibney) to the peculiar way in which a patient stands who has perinephritis, and this even when the inflammation has not reached the stage of abscess. The body is bent over to the affected side, the hip is a little flexed, and the hand rests on the same thigh. A certain resemblance to hip-joint disease is thus often assumed.

The urine is not necessarily affected; if the inflammation has resulted from ulceration of the kidney, pelvis, or ureter, pus from the perinephric abscess may pass into the urine (*pyuria*). In other cases albuminuria may occur from pressure of the abscess on the renal vein.

When pus forms, it is generally situated at first between the kidney and the lumbar muscles, and may make its way in various directions, unless evacuated by surgical means. If externally, it usually presents between the edges of the latissimus dorsi and the external oblique muscles; or it may pass downwards and point under Poupart's ligament. In other cases it opens into the colon, ileum or stomach; into the ureter, bladder, or vagina; or into the peritoneum, causing peritonitis. Or it perforates the diaphragm and sets up pneumonia, pleurisy, and empyema. Indeed, without perforation, it may cause pleuritic effusions, or merely compress the base of the lung by raising the diaphragm.

Diagnosis.—The conditions that are most likely to be mistaken for perinephritis or perinephric abscess are lumbago, spinal caries, cancer and tumours of the kidney, hydronephrosis and pyonephrosis, appendicitis, fæcal accumulations, splenic and hepatic tumours. The careful localisation of the lesion will distinguish it from spinal caries, hepatic and splenic tumours, and appendicitis. Fæcal accumulations, cancer of the kidney, and hydronephrosis, are not accompanied by fever; nor is lumbago, and this is often a bilateral trouble. The lateral inclination of the body in standing is a useful guide, but hardly serves in distinguishing it from hip-joint disease and psoas abscess.

Treatment.—This is mainly surgical. Local applications and opiates will relieve pain. If pus has formed it should be let out as soon as possible.

PYELITIS AND PYONEPHROSIS.

Ætiology.—Inflammation of the pelvis of the kidney, or pyelitis, arises from several causes, of which the following have been recognised:—(1) The action of turpentine and cantharides when given internally. (2) Febrile disorders, such as enteric and typhus fever, the exanthemata and pyæmia, as well as scurvy, diphtheria, and cholera. (3) It occurs, to a slight extent, in Bright's disease and diabetes; in the latter, probably as a result of the irritation of the saccharine urine. (4) Many cases are due to a definite local cause, such as the irritation of foreign bodies in the pelvis and infundibula of the kidney, especially calculi and gravel, but also hydatids, blood-clots, cancer, and tubercle. (5) Obstruction to the passage of urine may also lead to it by decompositions of the retained urine. (6) Inflammation may spread along the ureter to the pelvis, in cases of cystitis and other forms of inflammation in the urinary passages. It is somewhat doubtful how far cold may directly produce pyelitis. The causes under the fourth, fifth, and sixth heads are much the most frequent, and it will be seen that these include the usual causes of suppurating kidney, already described. Indeed, as already stated, pyelitis is constantly associated with that condition (pyelonephritis) or with cystitis (pyelo-cystitis).

Morbid Anatomy.—An acute or chronic form may be distinguished. In *acute pyelitis*, the mucous membrane is swollen, its vessels injected, and the surface covered with muco-pus; there are often small spots of hæmorrhage, and sometimes the inflammation takes on a diphtheritic form, patches of membrane adhering to the surface here and there. In calculous pyelitis, there may be ulceration of the surface from the presence of the stone; and such ulceration may, as already indicated, lead to perforation and perinephritis. *Chronic pyelitis*, in which the membrane has a white or ash-grey colour, is often the result of long-continued obstruction; and accordingly there is at the same time dilatation of the pelvis, infundibula, and calices, with flattening of the pyramids, and more or less atrophy of the renal structure. As a result of the pyelitis, pus and urine may accumulate to such an extent as to form a perceptible tumour (*pyonephrosis*); and the fluid so retained may from time to time, through a change in the obstructing conditions (*e.g.*, shifting of a calculus), be discharged into the bladder, so that the renal tumour subsides, and the urine suddenly contains a quantity of pus. Sometimes a pyonephrosis will open like perinephric abscess in various directions, such as into the loin, the iliac fossa, the bowel, the peritoneum, or the chest. In the renal pelvis there may be, besides pus and urine, blood, calculi, or other foreign bodies which have set up the mischief; and the urinary salts may be deposited, namely, urates in acid urine, and phosphates, if the urine is alkaline or ammoniacal, as it often is. The kidney is the subject of consecutive nephritis, either suppurative or interstitial, with more or less dilatation and atrophy. In some old cases the organ is so atrophied as to consist of little else than its capsule, and septa forming cavities which contain putty-like masses, the result of the inspissation of pus and the deposit of amorphous phosphates.

Symptoms.—In most cases the symptoms of pyelitis are combined with those of the lesions which have caused it. There is frequently some dull, aching pain in the loins, increased on pressure. The results of the inflammation generally show themselves in the urine. In early stages, there are some mucus, a few pus cells, epithelial cells from the pelvis and infundibula, and perhaps blood. The cells of the pelvic and infundibular epithelium are mostly conical, pyriform, tailed, or fusiform in shape. The urine is acid, and contains a mere trace of albumen.

In later stages, the urine contains pus in notable quantities.

Pyuria.—When such urine is passed it is turbid, and as it settles the pus forms a very pale yellow creamy deposit at the bottom of the glass, and mixes with the urine only just at the line of junction. The nature of this deposit can be determined by chemical tests, and by the microscope.

The usual *chemical* test is this:—The supernatant liquid is poured off, and some liquor potassæ or liquor ammoniæ is added to the deposit; it quickly loses its colour, becomes translucent, and changes into a viscid, ropy liquid, which falls from vessel to vessel in a more or less coherent mass. If the urine should decompose and become alkaline within the body, the pus will undergo the same ropy change, and the urine will be mixed with this viscid, glairy fluid, instead of with creamy pus. This happens sometimes in pyelitis and pyonephrosis, if the urine retained in the dilated pelvis at length undergoes decomposition; and it happens frequently from the same cause in cystitis.

Ozonic ether effervesces when added to pus, and may be used as a test for its presence in the urine.

Under the *microscope* the creamy deposit shows numerous pus-corpuscles—that is, more or less granular leucocytes, which, on the addition of dilute acetic acid, become clear, or display two, three, or four nuclei. In this stage, the epithelial cells of the pelvis are probably no longer produced.

The urine contains a very small quantity of albumen, derived from the liquor puris. It is often desirable to know whether the albumen present in such urine is solely due to pus, or is in part derived from diseased renal tissue. The pus should be separated as completely as possible by subsidence, or even by the use of the centrifuge. A large quantity of albumen must be due to something besides the pus, and the presence of casts would, of course, suggest that the kidney structure was involved.

It has been already stated that the purulent urine may be retained by an obstruction of the ureter: during such retention, the urine in the bladder will be quite normal; and it will again become purulent when the obstruction is partially or wholly removed. In such cases a tumour forms in the flank, consisting of the pelvis and kidney distended with urine and pus, so long as obstruction exists. It is more or less rounded, or oval, in some cases lobulated; lies between the costal margin and the crest of the ilium; is dull behind, and generally has the colon in front or to its inner side. There may be a line of resonance between its dulness and that of the liver and spleen, but the areas of dulness are often continuous. The tumour is usually painful and tender. It disappears for a time, if the obstruction gives way.

There is generally some febrile reaction in pyonephrosis, and rigors sometimes occur. Micturition is frequent in most of the severe forms of pyelitis.

Diagnosis.—Pyelitis in its early stage must be distinguished by the local signs, and the presence of the characteristic epithelium in the urine. When pus is contained in acid urine, it is more likely to come from the pelvis of the kidney than from an inflamed bladder. Pyonephrosis may be confounded with the numerous swellings which occur in the right flank, and which have been alluded to under Perinephric Abscess. Abscess and hydronephrosis are those which are most likely to give difficulty. In the former there is more pain than in pyonephrosis, more severe fever, fluctuation is more superficial, and the preceding hardness is less defined. The skin may be œdematous, and the urine is often free from pus.

The **Prognosis** depends very much upon the primary cause. Pyelitis following fever or a mild cystitis is likely to recover. Pyonephrosis is a serious lesion; it may be fatal by perforation into the chest or abdomen; by exhaustion from continued discharge, or by the induction of lardaceous disease. Rarely the pus inspissates, and a cure results with the loss of one kidney.

Treatment.—The primary condition must be studied, and removed as far as possible (*see* Tubercle of the Kidney and Renal Calculus). Where the pyelitis is more or less distinct from its cause, and open to separate treatment, this consists, in acute cases, of rest; dry-cupping of the loins if there is hæmaturia or much pain; the free drinking of warm bland liquids, by which the urine may be diluted; and the administration of salines (potassium citrate or acetate), with small doses of opium if there is much pain. When the case is more chronic and there is a free discharge of pus, astringents are commonly employed—namely, the mineral acids, alum, perchloride of iron, tannin, and acetate of lead, and, in very chronic cases, the balsams and allied drugs, oil of turpentine, oil of sandalwood, copaiba, benzoic acid, and cantharides, or direct antiseptics like salol. A general tonic regimen may at the same time be desirable, a nutritious diet, fresh air or the seaside, quinine and cod-liver oil.

If the distended pelvis forms a tumour (pyronephrosis), as a rule the operation of *nephrotomy* should be performed, and the cyst opened, especially if there be constant pain, severe fever, and interference with the action of the stomach and intestine; or if the tumour is increasing in size, inflaming the surrounding tissues, or threatening to rupture. Under opposite conditions, and if the cyst empties itself into the bladder from time to time, palliative measures may be tried, and Morris recommends rest in bed or on a couch, frequent hot baths, anodyne and emollient applications, gentle compression by belladonna plasters, and the avoidance of constipation and fæcal accumulation. The details of nephrotomy and the necessity for *nephrectomy*, or excision of the kidney, are questions for discussion in works on surgery.

HYDRONEPHROSIS.

By this term is meant the distension of the pelvis of the kidney by retained secretion; and the retention is, as a rule, the result of an obstruction in one or other part of the urinary passage, whether the ureter, the bladder, or the urethra.

Causes.—Hydronephrosis occurs at all ages, and is more frequent in females than males. It may be congenital—that is, in actual existence at the time of birth, when it may be so large as to constitute a serious obstacle to delivery; or it may develop after birth, although it is due to congenital causes; or it may be entirely the result of disease occurring in later life. Among the *congenital* causes are various abnormalities of the ureter, such as twists upon its axis, folds, reduplications, and valvular arrangements of the mucous membrane, contractions, or conversion into a fibrous cord. Sometimes the ureter joins the kidney at an acute angle, or the opening into the bladder is thick and rigid, or a branch of the renal artery lies across its course. Another cause is an imperforate urethra. Congenital hydronephrosis is often associated with other congenital defects and malformations, such as club-foot, hare-lip, or malformations of the external genitals; and those affected with it are frequently still-born, or live only a short time.

The causes *in later life* are all those kinds of obstruction which have already been referred to—namely, in the ureter, impacted calculus, cicatricial stricture, cancer of the abdominal and pelvic organs, ovarian tumours, and peritoneal bands; in the bladder, villous growths; and cystitis with hypertrophy of the walls; and in the urethra, stricture and enlarged prostate.

Anatomy.—Any long-continued obstruction leads to distension of the parts behind it. If it is in the upper part of the ureter, the pelvis dilates, subsequently the pyramids of the kidney become flattened, and the kidney undergoes those changes of chronic interstitial (consecutive) nephritis which have been already described (p. 811). If the obstruction is lower down—for instance, at the vesical orifice of the ureter—the ureter itself is involved in the distension. Such moderate degrees of hydronephrosis with consecutive induration of the kidney are common as the result of cancer of the uterus, vagina, and bladder in females. They, however, rarely lead to tumours that can be detected clinically, in many cases, no doubt, because death is brought about by the obstructive lesion too early for the extreme development of the hydronephrosis. But where the cause is congenital, or is less directly fatal in itself, such as calculus and strictures of the ureter, time is allowed for the full development of the pressure effects of the retained urine upon the renal structures. The pelvis becomes distended, and the kidney more and more flattened out; and finally a large cyst is formed, capable of containing forty or fifty ounces, or even several pints of liquid, and consisting of a thin membranous sac, which may present here and there portions of the kidney substance, but in some cases is quite destitute of any trace of it. Inside the sac there are sometimes septa dividing it into separate cavities. If the ureter is involved, it may be enormously dilated, to the size, perhaps, of the small intestine or colon, or it may be entirely merged in the distended pelvis and kidney.

The fluid of a hydronephrosis varies with the amount of kidney-substance still remaining; generally it is equivalent to a very dilute urine; it is pale yellow in colour, contains a small proportion of urea, uric acid and salts, occasionally a trace of albumen, or a little pus. Urea and uric acid are absent in some cases, and the fluid consists mainly of water with a trace of sodium chloride. Pus in any quantity is not present unless there has been previous inflammation of the pelvis, and the condition is then practically a pyonephrosis.

Hydronephrosis may affect one or both kidneys, according to the situation of the lesion or lesions causing it. In congenital cases it is frequently double—for instance, imperforate urethra must involve both kidneys equally; and the milder forms caused by cancer of the pelvic organs in women are often double. On the other hand, a calculus can only produce a single hydronephrosis, though it is possible that this cause might be in operation on both sides at the same time.

Symptoms.—A moderate degree of distension on one side, when the other kidney is healthy, may cause no symptoms whatever. If it is considerable, then a swelling is formed which becomes the prominent feature of the case, and has the usual characteristics of a renal tumour. It occupies one or other flank, extending from the costal margin to the crest of the ilium, and reaching, according to its size, towards the middle line, or even beyond it. A cyst holding forty or fifty ounces may cause scarcely any prominence of the abdomen, but its presence will be detected by resistance to deep pressure, and by the difficulty of bringing together the two hands, placed one in front, the other under the last rib. With larger cysts, an unsymmetrical enlargement of the abdomen is produced, and the loin and flank are bulged; or in extreme cases there is uniform distension similar to that of ascites or ovarian tumour. Sometimes the cyst is limited to the upper part of the abdomen, and may resemble enlargement of the liver by hydatid. The tumour is smooth or lobulated, in some cases tense, in others quite flaccid; and fluctuation can be sometimes obtained. It is dull on percussion, the dulness reaching back to the loin, and forwards perhaps nearly to the umbilicus; the colon lies in front of it, and may cause a resonant note.

An important feature of the tumour of hydronephrosis is its liability to suddenly diminish in size, or even disappear, from the escape of its contents into the bladder. Immediately afterwards, the patient has an abundant discharge of urine; and the cyst again slowly fills. Slighter variations in size or tenseness may occur, without any corresponding change in the urine being noticed. Local symptoms may be caused by the tenseness of the cyst, or its pressure on surrounding parts; such as pain, vomiting, dyspnœa, or interference with the heart's action. But these may be entirely absent. The urine in hydronephrosis is not much altered. Its quantity may be natural, since the healthy kidney compensates for the deficiency of its fellow. It may contain a trace of albumen, or a little pus; the urea and salts are in average quantity.

In cases of double hydronephrosis there is a tendency to uræmia from the retention of urinary constituents; the earlier indications of obstruction, when there is no tumour, are pains in the back or abdomen, partial suppression of urine from time to time, and increased frequency of micturition.

Diagnosis.—The tumour of hydronephrosis has to be distinguished from perinephric abscess, from pyonephrosis, hydatid of the liver or spleen, and, in extreme cases, from ascites and ovarian tumour. The history (*e.g.*, of calculus) may be the same in hydronephrosis, *pyonephrosis*, and *perinephric abscess;* the first is generally of longer duration, without severe, or even any, constitutional symptoms, whereas the other two conditions are likely to show evidence of suppuration. Perinephric abscess also gives the local signs of acute inflammation. In the absence of constitutional signs, pyonephrosis may be with difficulty diagnosed from hydronephrosis, and it may actually develop from it. *Hydatid* of the liver or spleen tends forwards or upwards, bulging the lower ribs, while hydronephrosis occupies the loin first; but I have seen a hydronephrosis due to calculus occupy the right upper quarter of the abdomen, just like hydatid of the liver. The disappearance of the tumour, coincident with an increased flow of urine, is in all cases the strongest evidence of hydronephrosis. *Ovarian* tumour should be recognised by the history of the enlargement, the position of the uterus, and the absence of the colon from the front of the cyst. In the rare case of a resemblance to *ascites*, the fluid withdrawn by paracentesis would contain urea and uric acid, and no albumen, or at most a mere trace, whereas in ascitic fluid albumen is abundant. In other cases also the aspirator and trocar may be used for diagnostic purposes.

Prognosis.—A single hydronephrosis may cause little or no trouble for many years; the kidney may gradually undergo atrophy without the distension being such as to cause any serious trouble. The risk in such a case is that the other kidney may be at some time involved (*e.g.*, by calculous obstruction), and then death may be brought about by uræmia. If the cyst reaches a great size, or becomes very tense, it may rupture into the peritoneum, or it may press on adjacent parts—the stomach and diaphragm—and cause death by interference with nutrition, respiration, or circulation; occasionally, a cure results from the spontaneous discharge of the contents of the cyst, which never again collect. In the case to which I have alluded, the fifth or sixth subsidence in the course of two years was accompanied by the discharge of a calculus, which became impacted in the urethra, and was removed by incision.

In double hydronephrosis, death results from the primary cause (*e.g.*, cancer), or from uræmic symptoms.

Treatment.—Since recovery from hydronephrotic tumour sometimes takes place by the pressure of the liquid overcoming the obstruction, an attempt should be made to get the same result by friction and manipulation of the cyst. But sometimes it is too painful, or too tense, to make such a course desirable. It may then be aspirated. Morris recommends that this should be done, on the left side, just at the anterior end of the eleventh intercostal space; on the right side, half way between the last rib and the crest of the ilium, and two inches behind the anterior superior spine of the ilium. The fluid will probably accumulate again, and then the next step should be incision and drainage of the cyst. At this operation a way may possibly be found into the bladder, and the aperture dilated, or an obstructing stone may be sought for and removed. Sometimes this results in complete cure, the secretion ceasing and the wound closing; in other cases a fistula remains, which is often quite manageable. If the discharge is a serious trouble, or if it becomes purulent and threatens to exhaust the patient, excision of the kidney may have to be discussed.

LARDACEOUS DISEASE OF THE KIDNEY.

(*Amyloid Kidney.*)

This has been generally classed as one of the forms of Bright's disease, although the essential change is not inflammatory.

The nature and ætiology of lardaceous disease have been already described (p. 687); phthisis, syphilis, continued suppuration, occasionally cancer and other cachectic conditions, produce the disease in the kidney as they do in other organs. But a slight amount of lardaceous change is found sometimes in cases of chronic nephritis, without any of the above causes being present; and it would seem as if nephritis itself might be a local cause of the degeneration.

Morbid Anatomy.—In the kidney the lardaceous change affects the vessels first. The glomerular tuft is often first altered, then successively the vasa afferentia, the vasa recta, the vasa efferentia, and the intertubular vessels. In some cases, however, the change can be found in the vasa recta before it is seen in the glomeruli. After the vessels, the basement membrane of the tubules is converted into the lardaceous material, but it is doubtful if the degeneration involves the epithelial cells of the tubules.

In the early stages of lardaceous change the kidneys present at first no appreciable difference, unless, perhaps, an undue distinctness of the glomeruli; but the application of iodine will bring out the vessels and Malpighian tufts by the dark brown or black colour (to the naked eye) which it gives them. If the change is very slight, it may be only observed in thin sections, stained and placed under the microscope. In advanced stages, the kidneys are much enlarged—it may be to twice their normal bulk. They have a whitish yellow colour, with some venules prominent on the surface; the capsule is slightly adherent. On section, the cortex is much widened, and has a pale yellow colour, more or less mottled with patches of white, while the pyramids are red or reddish-brown, forming thus a remarkable contrast to the cortex. The application of tincture of iodine produces a deep staining of the Malpighian tufts and their afferent arteries, and of the closely packed vasa recta in the pyramids, so that the vessels look as if injected. On microscopic examination, it is seen that there is, in addition, much evidence of inflammatory change, to which the enlargement of the kidney is to be in great part referred. The tubal epithelium is swollen, granular and fatty, the Malpighian capsules are thickened, and there are leucocytes extravasated into the intertubular tissue.

Sometimes the kidneys are almost normal in size, or even a little less, and somewhat rough or granular upon the surface.

Symptoms.—The symptoms of lardaceous degeneration commonly appear in patients already obviously suffering from phthisis, from tertiary syphilis with periosteal nodes, gummata, or necrosis, from caries, suppurating sinuses, discharging empyema, or similar lesions. Moreover, evidence of its involving the liver and spleen is sometimes, but not always, present when the kidneys are first called in question.

The first symptom is certainly sometimes the occurrence of albumen in urine otherwise normal—of average quantity, and good, even high, colour. With this there may be no other indication of renal disorder, only the evidence of the cause, and perhaps the changes in the liver and spleen. In other cases there is pronounced polyuria, and this may be the first change, even before the appearance of albumen (Dickinson). The urine amounts to seventy, eighty, or ninety ounces daily, it is pale in colour, of low specific gravity, 1008 to 1014, deposits little or nothing, and contains a varying, often small, amount of albumen. Paraglobulin is sometimes present in abundance, though this is not peculiar to lardaceous disease. In the deposit may be at most a few hyaline or granular casts, and, rarely, some that give the lardaceous reaction.

A third condition of the urine is seen in the last stage, when the lardaceous change is obviously complicated with nephritis; and the urine is scanty, highly albuminous, with numerous granular and fatty casts.

As to the cause of the alteration in the urine, it may be here enough to say that the degeneration of the walls of the vessels seems at once to account for the passage of water and albumen, as long as there is no great extent of tubal nephritis.

Next to the urinary changes, dropsy is the most frequent symptom. It has all the appearance of a renal dropsy, and there may be ascites and hydrothorax. Of its complications, pericarditis and peritonitis occur not infrequently, and may be fatal; but the other results of Bright's disease—namely, cardiac hypertrophy, high arterial tension, retinitis, hæmorrhages, and uræmia, are quite rare, though it appears that they do sometimes occur. Diarrhœa may occur from co-existing lardaceous disease of the intestine.

Death takes place from increasing dropsy, exhausting diarrhœa, serous inflammations, or from the effects of the original disease.

Diagnosis.—If there is a sufficient cause, such as phthisis, syphilis, or suppurating lesion, and evidence of lardaceous change in the liver and the spleen, which are generally enlarged, smooth, and hard (though they may be considerably diseased without increase of size), then the presence of albumen in the urine speaks strongly for lardaceous degeneration of the kidneys. All the more is this the case if the urine does *not* present the features of ordinary nephritis—that is, if it is of normal quantity and colour, or if, being abundant, it has a larger quantity of albumen than is common in granular kidney. If the condition of the urine is compatible with an acute or subacute tubal nephritis, lardaceous disease can only be inferred from the presence of an exciting cause and the evidence of the change elsewhere.

Prognosis.—This is decidedly unfavourable, especially if the stage of dropsy is reached. But in earlier stages, and in cases where the causative condition can be more or less controlled—for instance, tertiary syphilis by iodide of potassium, diseased joints and necrosis by suitable operations—the condition may last for some time without causing grave symptoms, and even it seems possible that practical cure may result.

Treatment.—This consists in removing the cause so far as it can be done. Beyond this the administration of iodide of potassium, of cod-liver oil, and of mild preparations of iron, such as the syrup of the iodide, seems to be attended with some benefit. Complications must be treated in the same way as in the different forms of Bright's disease.

CYSTS IN THE KIDNEY.

The following forms of cystic disease of the kidney occur:—(1) Small cysts in granular kidneys already described; (2) Extensive cystic change commonly known as "cystic disease"; (3) Simple cysts; (4) Dermoid cysts, which are exceedingly rare, and have the same characters as elsewhere; and (5) Hydatid cysts, which will be dealt with under Parasites in the Kidney.

Cystic Disease.

(*Conglomerate Transformation of the Kidney.*)

This occurs in adults and in the fœtus, and leads in both cases to an enormous enlargement of the kidneys. In adults, the organs weigh from one to five or six pounds, and may be detected during life as tumours occupying the lumbar region. They are more common in males than in females, and the patients are mostly of middle age or older.

Morbid Anatomy.—On section, the kidneys are seen to consist almost wholly of cysts of various sizes, containing a fluid which is clear, or turbid, yellow, pink, red, or purple, sometimes viscid, colloid, or purulent. Always albumen, and sometimes blood-discs, leucocytes, and cholesterin are found in the fluid, but urea and uric acid are generally absent. The cysts are surrounded by fibrous tissue, in which only remnants of renal tissue can be found; they are lined with epithelium. The pelvis, ureter, and bladder are healthy, or the pelvis may be dilated. The cysts undoubtedly arise from renal tubules which have been dilated as a result of obstruction; and are generally regarded as an extreme development of the cyst-formation which occurs in granular kidney. According to Bland Sutton, this disease may be regarded as a form of *adenoma*.

The **Symptoms** are often very obscure, but bear a general resemblance to those of chronic interstitial nephritis. The urine is generally abundant, of low specific gravity, pale in colour, and contains a little albumen and granular casts. Sometimes there is hæmaturia. Hypertrophy of the heart and high arterial tension have been observed. There may be lumbar pain, but dropsy is generally absent. In a patient under my care, there was daily intermitting pyrexia for several weeks preceding death. The termination is often by uræmic coma or convulsions; or there is cerebral hæmorrhage, suppression of urine, or pulmonary complication, such as bronchitis, œdema, or pneumonia.

For **Diagnosis** one must rely upon the presence of enlarged kidneys (for the change is generally double), associated with the characteristic urine, and cardio-vascular changes; but the disease is quite rare.

Treatment must resemble that of chronic Bright's disease.

In *Congenital Cystic Disease* the kidneys are greatly enlarged by cysts of the same kind as in the adult cases. They may occupy the greater part of the abdomen, pressing upon the diaphragm; and death may happen *in utero*, or during birth. The change is often associated with other congenital malformations, both of the urinary organs and other parts. Virchow supposed that the cysts arose by obstruction of the renal tubules *in utero* by uric acid infarcts. Shattock has advanced the view that they are due to a fault in the development of the kidneys; that the blastema from which the kidneys arise is not properly differentiated from that of the Wolffian bodies; that these bodies are mixed up, as it were, with the kidneys, and give rise to the cysts.

SIMPLE CYSTS.

Cysts of considerable size are sometimes found. They may be three or four inches in diameter, and exceptionally very much larger. They arise from the cortex, and project on the surface. Their contents are a clear limpid or gelatinous fluid, containing

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deposit, masses of lymphadenoma
angeioma, papilloma, sarcoma,
most common are sarcoma and ca
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SARCO

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children or infants. The organ is
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and inwards towards the umbilicus
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adrenal sarcomata is much

than in women. It is less common than secondary carcinoma. The cause of primary cancer of the kidney is not, as a rule, to be ascertained. Sometimes it appears to have followed a blow, or it is found associated with calculi; and it may be assumed, as in the analogous case of hepatic cancer and biliary calculi, that the calculi have acted as a constant source of irritation. It mostly affects one side only. It causes considerable enlargement of the kidney, but rarely to the same size, proportionately to the body, as does sarcoma in children. Encephaloid growths are most common, and colloid change frequently occurs, but scirrhus is rare. Hæmorrhage and softening often take place, as in cancers elsewhere.

Symptoms.—These are tumour, hæmaturia, and pain. The characters of the *tumour* have already been in part indicated; it may be irregular or nodulated; its mobility may be limited by adhesions; manipulation will elicit tenderness. Rarely the tumour pulsates, and a *bruit* may be heard in it. *Hæmaturia* is frequent; it is intermittent, variable in amount, but generally moderate. Albumen does not occur without blood, unless there is independent Bright's disease. Cancer-cells are sometimes recognised, but they may be lost among blood-corpuscles, or epithelium from the bladder or pelvis may be mistaken for them; and on the whole they cannot be relied upon for a diagnosis. Apart from blood and cancer elements, the urine may be perfectly normal in quality, density, and colour. *Pain* is variable, and often severe. It is situate in the loin and hypochondrium, or shoots down the groin to the thigh; it is not associated with retraction of the testicle. If blood coagulates in the pelvis of the kidney, the passage of the clots by the ureter may be accompanied by the pain of renal colic (*see* Renal Calculus).

The remaining symptoms of cancer of the kidney are anorexia, nausea, vomiting, irregularity of the bowels, either constipation or diarrhœa, and progressive emaciation and anæmia, as in other malignant affections. The duration varies with the kind of cancer—it may be from six months to two years, or even more. Death takes place commonly from exhaustion; but the growth may spread to the spine, and cause paraplegia; to the peritoneum and viscera, and cause local symptoms; it may rupture into the peritoneum; or there may be profuse bleeding into the tumour itself. Finally, secondary deposits in the brain may assist the fatal end.

Diagnosis.—Cancer of the kidney may be recognised by the co-existence of pain, hæmaturia, and tumour in the renal region; but, in the absence of hæmaturia, the nature of the tumour has to be very carefully investigated. It has to be distinguished, first, from enlargements of other organs; secondly, from other diseases of the kidney. From enlargement of the *liver* it is distinguished on the right side by the presence of a band of resonance between its upper margin and the ribs; if it is adherent to the liver, the edge of that organ may still often be felt on the anterior surface of the tumour. The presence of bowel (colon) in front of the tumour is also characteristic of its renal origin. The same holds good for the left kidney as compared with the *spleen;* and the latter is recognised by its sharp notched edge, its smooth surface, and its generally uniform enlargement in a downward and inward direction. An *ovarian* tumour grows up from below, whereas a renal tumour begins above and grows downward. *Mesenteric glands* lie nearer the middle line than the kidney, and form very nodular masses; isolated enlarged glands may give a clue to the nature of the larger mass. If cancer of the *colon* simulate renal cancer, intestinal symptoms are mostly present. *Accumulated fæces* on the left side would show more variability in size and consistence, and the diagnosis can be cleared up by the use of an enema. *Perinephric abscess* gives infiltration of the skin, local pain and tenderness, and febrile reaction.

The diseases of the kidney which may resemble cancer are pyonephrosis, hydronephrosis, hydatid, cystic degeneration, and tubercular disease. From the first three it should be distinguished by its solid feel; but a cancer may exceptionally be very soft, or a renal cyst may be small and tense, so as to give no certain sense of fluctuation. A tense uniform globular surface would be in favour of a cyst, but a lobulated hydronephrosis may be mistaken for new growth. By the time that *tubercular disease* causes enlargement enough to resemble cancer, it is practically a pyonephrosis. *Cystic* disease commonly involves both kidneys, and has not the other local signs of cancer; the pale abundant urine, with a trace of albumen, should distinguish it. In cases of doubt as between cancer and cystic enlargement, the aspirating needle may be safely used.

Treatment.—Cancer of the kidney is rarely recognised with certainty early enough for its removal by operation. The treatment must be mainly palliative; the objects being to relieve pain by opium and local applications, to keep up the strength by an easily digestible diet, and the moderate use of wine or small quantities of brandy.

TUBERCLE OF THE KIDNEY.

Tubercular disease of the kidney occurs in two forms:—(1) Primary disease, hitherto known as strumous pyelitis or pyelonephritis, and scrofulous pyelitis. (2) Secondary or disseminated grey tubercle.

PRIMARY TUBERCLE OF THE KIDNEY.

Ætiology.—This is obscure beyond the fact of its association with other tubercular lesions, and its probable dependence upon the same causes (hereditary influence, cold, deficient hygienic surroundings, &c.); but its localisation in the kidneys is not readily explained. It affects men more often than women, and occurs at all ages, but is least common in quite young children.

Morbid Anatomy.—The first change is the deposit of tubercles in the substance of the kidney, either cortex or pyramids. When first seen these are generally already yellow and cheesy; they enlarge, run together, break down into abscess cavities, and ultimately open into the calices and infundibula. But it is an important feature in the history of primary tubercle, that the kidney is not alone affected; the disease commonly affects at the same time more or less the urinary passages, and, it may be, every one of the urino-genital organs. Tubercles are deposited in the mucous and submucous tissues of the pelvis of the kidney, which ultimately break down, leaving ragged ulcers opening into the pelvis, and discharging pus, blood, tubercular *débris*, and portions of connective tissue into the current of the urine.

The *ureter* is similarly diseased; its wall is thickened and rigid from tubercular deposit, which finally ulcerates. The thickening may be sufficient to obstruct the canal of the ureter, or the latter may get blocked by fragments of tubercular matter, or by coagula of blood or pus coming down from the pelvis. If such an obstruction is at all complete, the urine and diseased products are retained in the pelvis of the kidney, distension takes place, and a pyonephrosis is the result. In extreme cases the kidney is almost entirely destroyed, partly by the spread of the caseating process, partly by the distension of the pelvis with retained products. It may be that scarcely any renal tissue remains. What has not been destroyed by tubercular ulceration is converted into dense fibrous tissue, and forms septa, separating the several abscess-cavities from one another; and such cavities may ultimately come to be lined with a smooth membrane. Their contents are a semi-fluid cheesy material, or a putty-like mass, which contains an abundance of lime-salts. The capsule is thickened, and may be even like fibro-cartilage.

The *bladder* is affected very like the ureter, either at the same time as or even before the kidney. Tubercle is deposited in the submucous tissue, and eventually the mucous membrane is covered with cheesy deposit, and much ulcerated. Sometimes the disease spreads into the *urethra*, and in men the *prostate*, *vesiculæ seminales*, and *testes* may become involved. The genital organs of women are much more rarely diseased under corresponding circumstances.

The disease may be unilateral, but more often affects both kidneys. It sometimes spreads to the second kidney from the bladder, and the ureter of the same side.

Symptoms.—In most cases the symptoms are those of pyelitis and cystitis combined, or pyelocystitis. There is more or less dull pain in the loins, which may be paroxysmal in character; and there is often some tenderness on pressure. Severe colicky pains occur if the ureter becomes plugged by tubercular *débris.* The kidney is not always large enough, or the pelvis sufficiently distended to form a tumour which can be recognised clinically.

The condition of the urine is most important. It is generally acid, with a more or less abundant deposit of pus, in which may be found pelvic and vesical epithelium, shreds of connective tissue, and *débris* of tubercle. With proper staining reagents after centrifugalisation tubercle-bacilli have been demonstrated, and should be looked for in all suspicious cases, as affording a certain means of diagnosis. Blood is often present from time to time, but not generally in large quantity. Albumen occurs in proportion to the amount of pus. Tube-casts are rare. The amount of urea will depend on the opposite kidney, which may hypertrophy enough to eliminate the normal amount. Sometimes the urine is ammoniacal and ropy, from retention and decomposition in the pelvis; sometimes from the co-existing cystitis. With cystitis, also, micturition becomes frequent and often painful.

Fever is generally present at some period of the disorder, and eventually becomes persistent, with morning remissions and high evening temperatures. As the disease progresses, emaciation, loss of appetite, and prostration become marked. In most cases other organs—such as the lungs and intestines—are involved. The opposite kidney also may be affected with the same lesion, or with lardaceous disease; and there is a gradual diminution in the excretion of urea, sometimes with scanty, occasionally with abundant, secretion of urine. Death takes place from the exhaustion of prolonged suppuration, from pulmonary or intestinal lesions, or from uræmia when the second kidney is seriously involved.

Diagnosis.—Tubercular kidney is most likely to be confounded with renal *calculus.* Hæmorrhage is more abundant in the latter, pus more continuously present in the former. Often the family history, the previous history of the patient, the co-existence of pulmonary phthisis, or other tubercular lesion, such as tubercle in the epididymis, suggests or confirms the diagnosis. Sometimes the ureter is so thickened by tubercle as to be felt through the abdominal organs as a hard rigid cord. Palpation of the kidney cannot be relied upon. The discovery of the tubercle-bacillus in the urine is conclusive.

Prognosis.—A favourable termination is in the highest degree improbable, chiefly because the disease is in most instances widely disseminated by the time its existence is discovered. The duration, however, is very variable.

Treatment.—The strength of the patient must be supported by good nutritious food, and by tonics, such as iron, quinine, cod-liver oil, and extract of malt; and opiates and local applications should be used to relieve the pain. Of late years this rather hopeless line of treatment has been supplemented by the surgical operations of nephrotomy and nephrectomy. The removal of the kidney can only be carried out where the disease is limited to one organ. It is out of the question, if already the other portions of the urinary organs are affected, or the kidney of the opposite side, or if tubercle has taken firm hold of other viscera; or if the patient is much exhausted. A small abscess in the kidney may be incised and drained, with some hope of ultimate success; and for large collections of pus in connection with obstructed ureters, nephrotomy is also valuable by relieving tension and pain, and giving direct exit to the purulent secretions.

SECONDARY TUBERCULOSIS OF THE KIDNEY.

This occurs as a part of acute general tuberculosis. The liver, spleen, and lungs are often at the same time affected. The tubercle appears in the form of minute grey or yellow deposits, one to two millimetres in diameter, scattered irregularly, and as a rule rather scantily, in the cortex and medulla of the kidney. A few may be seen on the surface, and others are revealed by section; they are round in shape, or slightly elongated in the direction of the tubules. They present the characteristic minute anatomy of tubercle. The rest of the kidney is healthy, and, as a rule, there are no clinical symptoms attending their deposition. Albuminuria may occur, and Roberts mentions a case from a French source, in which an unusually abundant deposit of tubercular granulations caused violent lumbar pains, with strong contraction and exquisite tenderness of the lumbar muscles.

PARASITES IN THE KIDNEY AND URINARY ORGANS.

The parasites invading the urinary organs are the *Echinococcus hominis* (a *hydatid*), the *Bilharzia hæmatobia*, the *Strongylus gigas*, and the *Pentastoma denticulatum*. The last two are exceedingly rare, and need not here be described.

HYDATID OF THE KIDNEY.

The life-history of the Echinococcus hominis and the development of hydatid cysts have already been described among the diseases of the liver. Hydatid cysts are very rare in the kidney; they form either in the substance of the gland, or between it and its capsule, and they grow to a variable size. They undergo the same changes as they do in the liver, and lead to corresponding local difficulties. As the cyst grows it gives rise to a tumour, which is generally globular and tense, and exerts considerable pressure on surrounding parts. Not infrequently it ruptures into the pelvis of the kidney, and the daughter-cysts, either whole or in fragments, escape and are discharged with the urine, if they are small enough to pass down the ureter. The cyst may rupture into the intestine, or it may, after compressing the diaphragm and the base of the lung, open into the bronchi. Such ruptures may occur spontaneously, or be brought about by a blow or other injury. In other cases, the cyst suppurates, or it becomes converted into the putty-like remains which have been previously mentioned (p. 694).

Symptoms.—One of the symptoms, and it may be the only one, is the presence of the tumour caused by the cyst. It is situate in the loin, with the colon in front of it; it is generally more or less globular and tense, and occasionally, but not always, gives the so-called hydatid thrill on percussion. The cyst may not be large enough to be detected, and may rupture in its early stage. If it bursts into the pelvis of the kidney, the urine will contain daughter-cysts or shreds of them, or a milky detritus, in which the characteristic hooklets may be found. The cysts, or any portions of them, may become impacted in the ureter, causing renal colic; or in the urethra, after passing through the bladder. If rupture takes place into the intestine or bronchi, cysts, or portion of them, will be got rid of by these passages, or the discharge of them may soon cease, and recommence after a longer or shorter interval. Pyelitis and cystitis may result from the passage of hydatids; but if no rupture takes place the urine is quite normal. Suppuration of the mother-cyst produces an abscess, which becomes manifest by increasing pain, tenderness, the implication of the surrounding tissues, and characteristic fever.

The **Prognosis** is fairly favourable, since a free discharge of the cyst and its contents is so often possible, and, as a fact, a good many recoveries have been recorded. The duration of hydatid of the kidney is variable, and may be as long as even thirty years.

The **Diagnosis** depends upon the presence of a renal tumour, combined with the discovery of cysts or hooklets in the urine. This latter is not in itself conclusive as to its position in the kidney, since a hydatid cyst behind the bladder may rupture into it. In doubtful cases a rectal examination should be made. A history of renal colic accompanying the discharge of cysts or fragments would be strongly in favour of their origin in the kidney. The tumour formed by the hydatid is most likely to be confounded with *hydronephrosis;* this may sometimes be distinguished by the typical variations of size; for, though a hydatid may also empty itself, this will be most likely accompanied by the appearance of daughter-cysts, &c., in the urine. But the resemblance may be so close as only to be solved by the use of the exploring needle, when the hydronephrosis will yield an urinous fluid, the hydatid a clear or opalescent fluid with a minute quantity of salts and a trace of albumen, and perhaps, under the microscope, some scolices and hooklets.

Treatment.—The simple withdrawal of the fluid by aspiration has yielded good results in the case of the kidney, as it has in the case of the liver. But a more certain method of cure is to cut down upon the tumour, through the loin if possible, to empty the cyst by tapping, and then incise it, and stitch the membrane to the edges of the wound in the parietes. If the contents of the cyst are being discharged by the bladder, this operation is not necessary. Impaction of cysts in the ureter requires the treatment proper to renal colic: opium, hot baths, and soothing local applications.

BILHARZIA HÆMATOBIA.

In different parts of Africa (Egypt, Natal) the inhabitants are liable to a peculiar form of hæmaturia, which has now been satisfactorily traced to the invasion by a parasite of the mucous membrane of the urinary passages. This parasite, called *Bilharzia*, from its discoverer Bilharz, belongs to the order *Distomidæ.* It is elongated in shape, the male being about half an inch, and the female three-quarters of an inch in length. In congress, the female lies in a groove on one side of the male, called the gynæcophoric canal. The ova are egg-shaped bodies, $\frac{1}{170}$ inch in length, and $\frac{1}{400}$ inch in thickness, and present at one end a sharp spine, which is sometimes quite at the extremity (terminal), sometimes a little distance from it (lateral). The embryo is a ciliated body, which can be seen moving within the ova, and, when liberated, glides rapidly about by the action of its cilia, and with various contractions of its body.

The parasites and the ova have been found in the minute veins of the bladder, ureter, pelvis of the kidney, uterus, and rectum, and in the portal vein and its tributaries; and, as a consequence, extensive inflammatory changes occur in the mucous membrane and submucous tissues of the bladder, ureter, pelvis, and rectum. The mucous membrane of the bladder, especially posteriorly, presents patches from a quarter to one inch in diameter, which are swollen, vascular, ecchymotic, and covered with tough mucus or yellowish exudation; or there are warty prominences encrusted with urinary salts. The ova of the bilharzia are found in great numbers in the mucus and exudation on the surface, and in the mucous and submucous tissues; and the parasites themselves lie in smooth-walled spaces, which are, no doubt, altered veins. In the ureter similar changes occur, and the swelling causes obstruction to the passage of the urine, and it may lead eventually to pyelitis and pyonephrosis. The parasite directly affects the pelvis of the kidney much less commonly. The ova may do harm in another way—namely, by serving as a nucleus for urinary calculi, which, indeed, appear to be quite common in Egypt. In the rectum, the parasite leads to polypoid growths, which may be mistaken for piles, and to large hæmorrhages under the mucous membrane. Some observations have shown that when the parasite invades the urinary organs the ovum is provided with a terminal spine, and when it attacks the intestine it carries a lateral spine.

There is some doubt as to how the parasite finds an entrance into the body. It is probable that there is an intermediate stage between the embryo seen in the human urine and the fully developed worm; and this half-developed creature may (as in the case of the sheep-fluke) inhabit the body of some mollusc. Leuckart suggests that the parasite is introduced into the system by the affected individual swallowing such molluscs while eating raw vegetables. Another possibility is that the parasite is taken into the stomach with drinking-water. J. Harley suggested that it might gain entrance to the body during bathing, either through the skin or through the orifice of the urethra; and this view is very strongly supported by Allen, of Pietermaritzburg, who points out the prevalence of this endemic hæmaturia amongst boys who bathe in the streams, and stir up the mud which forms their bed. He thinks that the parasites lodge in the prepuce, and in course of time find their way into the orifice of the urethra.

Symptoms.—These are hæmaturia, sometimes pain in the loins or perinæum, and, in severe cases, anæmia, from loss of blood. The hæmaturia is mostly of vesical origin; the urine is passed clear, and is followed by about a teaspoonful of blood. Allen says the blood is sometimes mixed with the urine; sometimes, on the other hand, there is a discharge of blood from the urethra on any considerable movement of the body. In such cases the urethra must be affected, and he states that he has felt, by means of the catheter, the obstruction due to the changes in the mucous membrane of that canal. The urine may at times contain no blood, but only whitish flocculent matter, and shreds and filaments of mucus in which the ova of bilharzia are found in great numbers. There is often a trace of albumen.

The symptoms may be of very temporary duration, and boys seem often to get quite well in a short time. But the loss of blood may lead to a high degree of anæmia, and, if the inflammatory changes are severe, there may be all the symptoms of pyelitis and pyonephrosis, with secondary septicæmia. But in many cases the vitality of the parasite seems very great and the patient suffers from an intractable cystitis.

Treatment.—Harley states that the oils of turpentine and male fern given together in 15-minim doses with 5 minims of chloroform and tragacanth mixture every morning brought away great numbers of the ova. He also recommends the internal use of bicarbonate of potassium, and the injection into the bladder every second or third day of five ounces of tepid water containing twenty or thirty grains of iodide of potassium. This proceeding seems, however, to have set up cystitis in at least one case; and Cobbold objected strongly to the use of anthelmintics, diuretics, or injections. He recommended a tonic treatment, the use of infusion of buchu, a liberal diet, and gentle exercise; but he did not look for a cure until a long time had elapsed. Manson also regards parasiticides as useless or harmful, and recommends the disease to be treated as a chronic cystitis, by washing out the bladder with weak boric acid lotion, and by the internal use of diluents, of uva ursi, buchu, &c., and of anodynes when necessary. Polypoid growths in the rectum should be removed.

Prevention.—The drinking-water should be from a pure source and should be filtered, and uncooked vegetables, fish, and molluscs should not be eaten. Allen, in accordance with his view of the manner of entry of the parasite, recommends avoidance of bathing, and even suggests circumcision.

MOVEABLE KIDNEY.

The kidney normally extends from the lower border of the eleventh dorsal vertebra to the lower border of the second lumbar vertebra, its inner margin being, on an average, three inches from the middle line; and it is fixed in this position by its surrounding capsule of adipose tissue, by the layer of peritoneum in front of this, and by the renal vessels. The name *moveable kidney* is given to one that is readily displaced from its normal position, and can be moved more or less freely in the abdomen. This unusual mobility may be congenital or acquired.

Congenital mobility is due to the presence of a *mesonephron*—that is, the kidney is partially or completely surrounded by peritoneum (like the colon), and is thus free to move about among the abdominal viscera. This condition is quite rare. It is sometimes distinguished as *floating* kidney.

Acquired mobility is much more common. It affects females more often than males; and the right kidney becomes moveable thirteen or fourteen times as frequently as the left. Sometimes both are affected at the same time. The age of the patient is mostly between twenty and fifty. It mainly results from conditions which stretch or relax the tissues and structures surrounding the kidney, especially the fatty capsule and the peritoneum. Perhaps the most frequent cause is repeated pregnancy, by which the peritoneum is dragged upon and stretched, and afails fter delivery to recover its normal tension. But moveable kidney is not confined to those who have borne children. Emaciation by reducing the fat surrounding the kidney may be a cause sometimes. Many patients have a pendulous abdomen, and general want of tone in the abdominal and pelvic tissues (*Enteroptosis, see* p. 718). It is asserted by some that recurring congestion of the kidney, in association with menstruation, may lead to displacement. At any rate, an increase of size of the kidney from any cause must favour it. Tight lacing has been charged with it, but it frequently occurs independently.

Symptoms.—The most common subjective symptom is a sensation of weight, or dragging, or pain in the loin or side of the abdomen affected ; and this may be constant, aggravated by walking or exertion, and relieved by lying down. From time to time there may be severe attacks of so-called *strangulation* of the kidney, consisting of great pain and tenderness in the renal region, with scanty, high-coloured, and even bloody urine. There may be nausea or vomiting, and malaise; but generally not much pyrexia. Such an attack, which subsides in the course of a week or more, is probably due to twisting or kinking of the renal vessels by the movements of the kidney.

Rarely does a moveable kidney produce any considerable pressure on surrounding organs, for its very mobility renders this unlikely; but œdema from pressure on the inferior vena cava has been recorded. Some gastric disturbances, such as nausea, flatulent distension, &c., are probably due rather to the nerve-connections of the kidney and of the stomach than to pressure on the duodenum, as suggested by one author. Lastly, many patients suffering from displaced kidney are nervous, hysterical, or hypochondriacal.

The evidence of moveable kidney lies in its detection by palpation of the abdomen. In the majority of cases it is felt only in the flank of the side affected. Here a smooth, firm but not hard, rounded tumour may be felt, of about the size of the kidney. If it lies between the last rib and the crest of the ilium, it can be pushed more or less in all directions, but most easily upwards towards the thorax, when it may get entirely out of reach, leaving the flank normal. Often when the patient lies down, nothing is felt until she takes a deep breath, when the kidney glides down, and may be secured by dipping the fingers in above its upper extremity. If the organ be pressed firmly, or grasped, the patient experiences a sharp pain, or sickening sensation. The examination should be made with both hands, one pressed firmly in between the last rib and the crest of the ilium, the other on the front of the abdomen. In some cases the kidney rises towards the front of the abdomen, or rests in the iliac fossa, or can be pushed over towards the middle line or beyond it. It does not seem that any difference in this respect exists between the kidneys that possess a mesonephron and those that do not; at any rate the latter often acquire a very considerable degree of mobility.

Landau lays much stress upon hydronephrosis from repeated kinking of the ureter as a possible result of this complaint; except for this, mobility of the kidney is rather a discomfort than a serious disease, and does not tend to a fatal result.

Diagnosis.—A moveable kidney is likely to be confounded with small ovarian cysts, with distended gall-bladder, with tumours of the omentum, mesentery, stomach, pancreas, ascending or descending colon, with retained fæces, or with enlarged spleen. *Ovarian tumours* are moveable only in directions determined by their pelvic connections, and cannot be pushed up into the loin. A *distended gall-bladder* has dulness continuous with that of the liver, but may be extremely moveable. Its cystic nature may be recognisable. *Tumours of the alimentary canal* or *pancreas* have rarely a perfectly smooth surface, vary in shape and size, and are more continuously painful. An enlarged *spleen* is never behind the intestines; the lower it lies, the more it gets to the front of the abdomen, and it is always close under the parietes.

Treatment.—The object of treatment, if symptoms are severe enough to require it, is to retain the kidney in its normal position, and so prevent the weight, dragging, and pain, as well as the strangulation and other symptoms that may result from kinking of the vessels of the ureter. Abstention from violent exercise may be enough in some cases, and rest in the recumbent posture will at all times give relief. But as getting about again brings on the troubles, it is desirable to attempt support of the kidney by some kind of pad, truss, or bandage. A spring truss, with a large pad pressing on the front of the affected loin, may be used, or a broad bandage with a large pad sewn into it, and so arranged as to prevent vertical displacement. Dr. Newman, of Glasgow, recommends a tight-fitting elastic bandage, extending from the groin to the sixth or seventh rib, under which an air-pad, capable of being inflated while in position, prevents the descent of the kidney. The treatment of the symptoms of strangulation consists in complete rest, the use of poultices and hot fomentations to the loin and abdomen, and opium or morphia by injection or suppository. Severe cases justify the operation of *nephrorrhaphy*, by which an incision is made in the loin and the kidney is stitched to the parietes. It may be even desirable to excise the kidney altogether if nephrorrhaphy fails, and especially if the kidney is diseased as well as moveable, with the proviso that the other kidney is sound.

RENAL CALCULUS.

(*Nephrolithiasis*).

The following are the varieties of urinary calculi. The first five are the commoner forms ; the others are much more rare :—

1. *Uric Acid.*—Hard, round or oval in shape, smooth or finely tuberculated, sometimes facetted from contact ; of yellowish, fawn, or reddish colour. They vary in size, from that of poppy-seeds to that of mustard-seeds or peas, and are occasionally very much larger. Frequently they exist in great numbers.

2. *Sodium Urate.*—Soft, not generally of large size.

3. *Calcium Oxalate, or Mulberry Calculus.*—These are very hard, rough or irregular on the surface and of blackish-brown colour ; when smaller they are smooth, rounded, greyish or brown in colour. Generally they are solitary.

4. *Mixed Calcium and Ammonio-magnesian Phosphate, or Fusible Calculus.*—The mixed phosphates are precipitated in urine rendered alkaline by ammoniacal decomposition, such as occurs when the secretion is retained in the bladder or in a dilated pelvis. They rarely form the nucleus of a stone, but are deposited upon other calculi of uric acid or oxalate, upon foreign bodies (*e.g.*, in the bladder), and upon the inflamed mucous membrane of the bladder or of the renal pelvis. They may thus enormously increase the size of vesical stones, and in the pelvis may form concretions, which are moulded to all the infundibula and calices. The deposit is white, soft, friable, and fuses under the blowpipe into a kind of enamel.

5. *Calcium Phosphate.*—White and chalky, rather smooth on the surface, with an earthy fracture, varying in size from that of a pea to that of a hen's egg.

6. *Calcium Carbonate.*—Small, very hard, smooth, grey, yellowish or bronze-coloured, and varying from minute grains up to stones the size of a hazel-nut.

7. *Cystine.*—Usually egg-shaped, the surface granular, glisten-

ing with crystals of yellow colour, looking translucent on section, with indications of a radiating structure, and rather soft in consistence. They become green on exposure. With a lens the hexagonal form of the crystals may be seen.

8. *Xanthine.*—In physical characters like uric acid calculi, but of a cinnamon colour, soluble in liquor ammoniæ and liquor potassæ. They are extremely rare, and have not been found in the renal pelvis.

9. *Urostealith.*—Soft greasy concretions, which have been found in a few cases; one was shown to consist of about one-third cholesterin and fat, one-third uric acid, and some oxalates.

10. *Indigo.*—Once found in the renal pelvis by Ord as a calculus weighing 40 grains.

The majority of calculi consist of more than one of the above substances, and sometimes there are found alternating layers of uric acid, oxalate, and phosphates, laid upon one another under varying conditions of the urine. Phosphates and carbonates are deposited in alkaline urine; the remainder of the calculi above enumerated form in acid urine.

Urinary calculi vary much in size; they may be two or three inches in diameter, or they may consist of very small particles, and are then known as *gravel.*

For long it has been believed that the centre or nucleus (*i.e.*, the first-formed portion) of most calculi is uric acid; but even within that, calcium oxalate or sodium urate has been found. Some calculi are deposited upon a nucleus of blood-clot, mucus, or renal casts, and the ova of Bilharzia hæmatobia may form the starting-point of renal stones. Most calculi are formed in the urinary tubules, and some even in the epithelial cells (Ralfe); and the frequency of uric acid and calcium oxalate as components of stone is, no doubt, determined by their relative insolubility. Another important factor seems to be the presence of some "colloid matrix," such as may be formed by mucus, blood, or perhaps the protoplasm of the epithelium-cell; since it has been shown by experiment that in the presence of viscid solutions a chemical precipitation does not take place rapidly in a crystalline form, but more slowly in the shape of granules, spheroids, and laminæ, which has been called *submorphous.* Such colloid matter, therefore, determines the form of the precipitated matter, and may also bring about the precipitation in a secretion overcharged with the relatively insoluble salt. Ralfe thinks that the cause may often be an impairment of vital power in the renal cells, so that they fail to secrete uric acid or oxalate, and hence these substances are actually deposited in the cells, the cell substance acting as "colloid." Ultimately the calculus grows by accretion of other deposits upon it. Roberts shows that the precipitation of uric acid from the urine is favoured by great acidity of the urine, by excess of uric acid, by decrease in the urinary pigment, and by decrease in the urinary salines; and that it is retarded by the opposite conditions.

Ætiology.—Calculus is very much more frequent in the eastern part of England than in the middle or western parts; a difference which is not at once explained by the existence of a chalky soil in the former, since the majority of calculi from patients in these parts are of the uric acid variety. Stone is more common among the poor than the rich, in males than in females, and perhaps more so in early or late life than middle age. Ralfe points out that these are periods of vital impairment. For instance, febrile illnesses are common among children, and many suffer from malnutrition; on the other hand, in old age, besides the obvious waning of the powers generally, there is often local impairment from urethral stricture, or prostatic enlargement, or diminished expulsive powers.

Symptoms.—(1) When the stone exists in the pelvis of the kidney, it may remain entirely latent, or it may give rise to lumbar pain, hæmaturia, albuminuria, or the passage of pus from the induction of pyelitis; and these changes may result from the presence of very small stones, or of the deposit known as gravel. (2) If it falls into the ureter it may become impacted or move along with great difficulty, causing *renal colic*, hæmaturia, and, under certain circumstances, *obstructive suppression*. (3) The later effects of real stones, either in the kidney, or after impaction in the ureter, are pyelitis of all degrees, pyelonephritis, perinephritis, perinephric abscess, hydronephrosis and pyonephrosis, the symptoms of which have been already described.

Gravel and calculus are frequent causes of *lumbar pain*, which is often regarded as "lumbago" or muscular rheumatism. The lumbar pain or aching may be rendered worse by jolting or shaking. If the symptoms are of long duration, albumen, pus, or mucus may be passed, and from time to time blood in varying quantities. Occasionally, small calculi or gravel are discharged with the urine. The subsidence of symptoms entirely after a long period of activity may be due to the calculus becoming encysted.

Renal colic is caused by the spasmodic contraction of the muscular fibres of the ureter, irritated by the passage of the calculus. It is comparable with biliary colic, and is characterised by intense pains, rigors, nausea, and vomiting. The pain is situated in the loin and flank of the same side, and radiates downwards and inwards to the groin and testicle; sometimes to the thighs, and even to the heel and sole of the foot; at others to the abdomen, chest, and back. In the severer attacks the patient is doubled up with the pain, or writhes on the floor, and bursts out into profuse perspiration, or he becomes pale and collapsed, with quick, feeble pulse; but the temperature may be raised. With this there are nausea and vomiting, often a rigor, and sometimes even general convulsions. The testicle on the same side is retracted, and is swollen and very tender. The pain may be less for a time, but soon returns, and altogether it may last a few hours or a day or two, until at length the stone is passed into the bladder or returns to the pelvis, when there is sudden relief, and only an aching, smarting sensation in the side is left. The pain may, however, cease when the calculus still remains impacted in the ureter. During the attack, micturition is frequent and painful, and the urine is scanty, coming, perhaps, only by drops; and it may contain blood. By examination of the abdomen, one can sometimes detect the stone in the ureter, and watch its course from kidney to bladder.

Such an attack may occur spontaneously, or may be brought on by some movement which appears to dislodge the calculus from its position. Renal colic may recur in the same patient. This, of course, must depend on the number and size of the stones; obviously, if a stone gets back into the pelvis it may set up renal colic on again becoming impacted.

Obstructive suppression is distinguished by Roberts from the suppression which results from acute congestion or acute inflammation in Bright's disease, and the symptoms in marked cases are strikingly different. Obstructive suppression arises when both ureters are simultaneously compressed, as occurs in women when cancer of the pelvic organs invades the base of the bladder; or when one kidney has been disorganised, or excised, or otherwise placed *hors de combat*, and a calculus becomes impacted in the ureter of the healthy organ. In some cases, no urine is voided; in others, a certain amount may be passed in small quantities at long intervals, but it is clear, watery, of very low specific gravity —*e.g.*, 1006—and contains an extremely small quantity of urea or other solids; and there is no albumen, unless there is blood, or unless the urine is modified by the cystitis which accompanies cancer of the bladder. The patient's condition is not at first materially altered. He may eat as much as usual, but he loses muscular power, and becomes sleepless, and after some five or six days he is seized with muscular twitchings or jerkings, affecting the arms, legs, and trunk. The pupils are contracted, the temperature of the body falls, the breathing is slow, panting, and laborious, the mouth and tongue are dry, and there is great thirst and there may be troublesome vomiting. The muscular twitchings continue, and the patient becomes restless, indifferent, and drowsy, but neither convulsions nor coma occur. Death, as a rule, ensues from nine to eleven days after the commencement of the obstruction, and is very rarely postponed beyond this. Recovery may take place if the obstructing calculus is passed or dislodged from the breaking down of any new growth that has been the means of blocking the ureter. Roberts points out that an obstructive suppression of a less dangerous kind may arise in the course of a double hydronephrosis. Bradford shows that symptoms identical with the above may arise when the functions of the kidney, hitherto healthy, are disturbed by distant lesions, presumably acting reflexly, or by toxic influence.

The production of pyelitis, hydronephrosis, and perinephric abscess as a result of stone, and the symptoms indicating them respectively, have already been discussed.

Diagnosis.—The typical symptoms—lumbar pain, hæmaturia, and albuminuria—may be caused not only by a medium-sized calculus, but also by fine gravel and uric acid crystals, which will readily pass the ureter; and in these cases testicular pain and frequency of micturition may also be present. The diagnosis may be made by rendering the urine alkaline, when the symptoms will quickly diminish or cease altogether. A severe attack of colic, associated with hæmaturia and testicular pain and retraction, is very strong evidence of calculus; but other foreign bodies besides calculus may set up colic while passing the ureter—*e.g.*, masses of viscid mucus the result of pyelitis, clots of blood which have formed in the renal pelvis, fragments of tissue in cancer of the kidney, and, exceptionally, hydatid daughter-cysts.

Some differences in the character of the hæmaturia depend upon the *seat* of the hæmorrhage (*see* p. 788).

Any disease leading to hæmorrhage from the kidney may for a time be mistaken for calculus, since hæmaturia may occur without other symptoms being prominent. In *cancer* of the kidney hæmorrhage is often more abundant and more continuous. Gelatinous red lumps appear in the urine after the blood; and sometimes cancer-cells may be found by the microscope. The presence in due time of a tumour will help the diagnosis. Calculus is more likely in a young patient, but either may be present in middle or old age. *Tubercle* of the kidney may closely simulate calculus, by lumbar pain, frequent micturition, pus in the urine, and even blood. In the former there may be a family or personal history, or present indications of tubercle; hæmaturia and renal pain are less prominent and characteristic. *Cystitis* is simulated by the frequency of micturition which occurs in renal calculus, especially if hæmaturia is absent. Previous attacks of lumbar pain, and the acid reaction of the urine, if pus is present, are in favour of a renal origin. *Hysteria* may simulate renal pain, and requires very careful consideration.

On rare occasions, when the renal pelvis contains numerous stones, these may be recognised by a crackling sensation conveyed to the fingers placed under the twelfth rib; and a diagnosis is sometimes effected by the surgeon making an exploratory incision, and sounding the kidney with a long needle. In a few instances the presence of a stone in the kidney has been demonstrated by the use of the Röntgen rays, or Skiagraphy: and in doubtful cases this should be used.

Some differences have been noted in the symptoms dependent on the chemical nature of the calculus. *Uric acid* calculi produce the least pain, which is dull and oppressive, with a sense of weight. The urine is acid; bleeding is frequent, not excessive, and periodic, apart from exertion. The mucous deposit is yellowish, or rusty. *Calcium oxalate* causes acute pain referred to a particular spot, as well as shooting in the ureter, shoulder, or epigastrium. The urine is less acid, bleeding is abundant, and the mucus is glairy and greenish. *Phosphates* cause severe unremitting pain, attended with exacerbations. The urine has been at some time alkaline, there is much muco-pus or flocculent mucus, which may be tinged with blood, though free bleeding is rare.

Treatment.—The frequent occurrence of gravel, which is nearly always uric acid, should be met by the administration of drugs to render the urine less acid, or even alkaline. The most efficient are the citrate, acetate, and bicarbonate of potassium, which may be given in 30 or 40 grain doses in 3 or 4 ounces of water two or three times daily, or in one larger dose before the night's rest, during which time the tendency to uric acid precipitation is greatest (Reberts). Pure or distilled water may be also drunk, and the diet should consist largely of farinaceous and vegetable food. On the other hand, alcoholic liquors and highly nitrogenous food should be avoided. When, in addition, lumbar pain and a trace of albumen suggest that the precipitation is taking place in the kidney, the same line of treatment may be pursued, and so long as the deposit is only minute it may be washed away by the urine, and the alkalisation of the urine will prevent any more being formed. Ralfe recommends turpentine in capsules every morning or twice daily. If the existence in the kidney of an actual calculus is presumable from the occurrence of one or more attacks of renal colic, this "solvent" method of treatment may be persisted in. The dose of the acetate or citrate of potassium should be 40 or 60 grains every three hours, continued for some months. The treatment may be assisted by occasional saline purges. Benefit is often derived from residence at Vichy, Ems, Carlsbad, Salzbrunn, Tarasp, Neuenahr, or Wildungen, where the waters are alkaline or saline. As a preventive of stone exercise is desirable; but the encystment of a large calculus is likely to be favoured by rest in the recumbent position, inclining to the affected side.

Treatment of Renal Colic.—Anodynes are here required, both to relieve the intense pain, and because they may also relax the spasm of the ureter, and so facilitate the escape of the stone. If the pain is severe, a morphia injection should be at once given; or morphia or opium may be given internally, or in suppositories, or chloroform or ether may be inhaled. Locally, hot poultices, hot fomentations, belladonna applications, or the hot bath should be used. The patient should be at rest, and warm diluent drinks, barley-water, &c., should be taken from time to time.

Surgical Treatment.—When the symptoms of stone in the kidney are very severe, and remain unrelieved after prolonged medical treatment, or if the patient is prevented from following his livelihood, an operation for the removal of the calculus (nephro-lithotomy), or even of the kidney itself if the calculus cannot be found, may be judged expedient.

MORBID CONDITIONS OF THE URINE INDEPENDENT OF DISEASE OF THE URINARY ORGANS.

DIABETES INSIPIDUS.

It has been already shown that polyuria, or excessive secretion of urine, may arise from various morbid conditions, such as chronic granular kidney, lardaceous disease of the kidney, and hysteria. But there are two forms of polyuria, which have long been known under the equivalent term diabetes (διαβαίνω = I go through), and which require special description. In the more common form, *diabetes mellitus*, sugar is present in the urine at the same time; in the other form, there is no sugar, and it is called *diabetes insipidus*.

Ætiology.—Diabetes insipidus is a comparatively rare complaint. It occurs mostly in early adult and middle age, but sometimes in quite young children, and it is more frequent in males than in females. It cannot be always traced to a definite cause, but it has occasionally followed upon blows or injuries to the head, emotional disturbance, or convalescence from acute diseases. Family predisposition has also been recorded, the disease being handed down from parents to children. A nervous origin is suggested by the above, and this is supported by the knowledge of the experiment of Bernard, who produced polyuria by puncturing the fourth ventricle at a point a little above the centre for the production of sugar in the urine. Tumours of the brain, also, now and then produce polyuria, but it is rarely that cases of pronounced diabetes insipidus have been shown to be due to them.

Symptoms.—These begin either insidiously or suddenly; sometimes they have followed immediately upon the ingestion of a large quantity of water. The prominent symptoms are the enormous quantity of water passed, and the great thirst by which the patient is led to replace the loss. The urine may reach fifteen, twenty, or even forty pints in the twenty-four hours. It is very pale, almost like water, of specific gravity 1002 to 1005, and faintly acid in reaction. The percentage of solid constituents is, of course, small, but the daily excretion of urea may be somewhat above the normal. Sometimes *inosite*, or muscle-sugar, has been found; but it is not invariably present, and it has also been detected in some cases of diabetes mellitus and of Bright's disease. Quite exceptionally, a trace of albumen is present, and more often minute traces of grape-sugar. The thirst is excessive and uncontrollable, the patient being obliged to drink large quantities of water to replace what passes by the kidneys.

Other symptoms are the following:—The mouth and tongue are usually dry, the skin is dry, and the temperature is normal. The appetite may be unaffected; sometimes, however, it is enormous, as it is so often in diabetes mellitus. The bowels are regular, or only slightly constipated. Beyond this, the patient may be in the enjoyment of very good health, and he finds the diabetes an annoyance rather than an illness. But often, especially in the severe cases, there are emaciation, weakness, and languor; the sleep is much disturbed, and there is mental depression or irritability of temper; occasionally the sexual powers are abolished.

The course of the disease is variable. If it arises from injuries to the head, it may be of short duration; when it is due to cerebral tumours, it will probably terminate in death in the course of a few months. Spontaneous and idiopathic cases may last for years, and are most intractable. They are rarely fatal, except from the intervention of other illnesses, especially phthisis and pneumonia.

Morbid Anatomy.—The lesions which are at all constantly found in diabetes insipidus are very few. Dilatation and hypertrophy of the bladder, dilatation of the ureters, and enlargement of the kidneys may be seen, and are attributable to the prolonged pressure of large quantities of urine. Besides, there are found, in some cases, the lesions of the intercurrent diseases which have proved fatal, such as phthisis or pneumonia; in others, the cerebral lesions, which may have been the primary cause. As to the nature of the disease, Fagge suggests that it is due to dilatation of the renal vessels from loss of controlling action of their vasomotor nerves. It clearly has a nervous origin.

Diagnosis.—This is not generally difficult. The enormous quantity of pale urine, of low specific gravity, without abnormal ingredient, and the accompanying thirst, are distinctive. But care must be taken to exclude other forms of polyuria, such a those from *Bright's disease* and *hysteria*. In the former there i generally at some time or other a distinct trace of albumen, th quantity of urine is not so considerable, and other indications ar present, such as high arterial tension and cardiac hypertrophy In hysteria, the condition is but temporary. The urine of *diabete* *mellitus* is at once distinguished by its higher specific gravity an by the presence of grape-sugar.

Treatment.—The removal of the cause is rarely possible, an the treatment generally resolves itself into attempting to influenc the nervous system by certain drugs. Valerian is highly spoke of by Trousseau and others. It should be given at first in 5-grai doses of the powdered root three times a day; the dose should b increased by 5 grains at a time, and, if necessary, to the extent o 2 or 3 drachms in the day. The infusion or extract of valeria and valerianate of zinc (up to 20 grains daily, in divided doses may be substituted. Ergot, ergotine, codeine, bromide of arsenic carbolic acid, sodium salicylate, and dilute nitric acid have als been tried; and, recently, good results have been recorded wit antipyrin in 8-grain doses every two or three hours, so as to reac 60 or 70 grains in the day. Galvanism is recommended in one o the following methods. The constant current is applied over th medulla oblongata or upper part of the spinal cord; or one pole i applied to the loin, and the other to the hypochondrium on the on side for a few minutes, and the same on the opposite side; or th anode is applied to the nape of the neck, and the kathode first t the loins and then to the epigastrium (Külz).

DIABETES MELLITUS.

The characteristic feature of this illness is the passage of larg quantities of urine containing glucose or grape-sugar.

Ætiology.—It is much more frequent than diabetes insipidus but has a somewhat similar causation. Thus, though in man cases the origin of the disease is quite unknown, in a certai proportion it can be traced to disturbances of the nervous system and it may be hereditarily transmitted. Among the forme causes are especially mental anxiety, emotional excitement, an occasionally injuries to the head; and it has been seen in association with exophthalmic goître. Other apparent causes ar exposure to wet and cold, acute diseases, malarial poisoning gout, rheumatism, and undue indulgence in starchy and saccharine food, and in alcohol. It occurs twice as frequently in male as in females. It is rare in childhood, but occurs at puberty, an most often in middle life and advancing age. It is more frequen in fair than in dark people; and it appears to be more prevalent in urban and manufacturing than in rural districts (Roberts), and in the eastern than in the western counties of England. Considerable differences are also noted in its prevalence in various parts of the world.

The Condition of the Urine.—The actual quantity of sugar in the urine in cases of diabetes varies from a mere trace up to a maximum of forty grains to the ounce. From eight to ten grains per ounce is the more usual amount. The daily excretion is often 6,000 to 7,000 grains. The presence of sugar is commonly accompanied by other changes in the urine. Its quantity is increased to ten, fifteen, or twenty pints per diem, and the specific gravity is raised by the presence of so much sugar to 1035, 1040, or 1045. It is said to have been even 1060 or 1070, but this must be rare. The colour of the urine is generally pale yellow, or almost like water; it has a sweetish odour like hay, and a sweet taste. The reaction is acid. If it contains much sugar it will leave a crystalline deposit as it dries upon linen or elsewhere. The urea of diabetic urine is in excess of the normal, sometimes very much so; uric acid is either unaffected, or, according to some, is below the normal. Phosphates and sulphates are usually in proportion to the urea. Ammonia also is said by Stadelmann to be present in large amount, but in spite of this the acid reaction is maintained by the presence of a new acid, β-oxybutyric acid. If this acid undergoes oxidation it is converted into aceto-acetic acid, which breaks up into carbonic acid and acetone. Thus, at different times in the course of diabetes, the urine may contain one or more of these three compounds, β-oxybutyric acid, aceto-acetic acid, and acetone. If a solution of the perchloride of iron be added to diabetic urine it will frequently give a rich claret-red coloration, which is at present believed to be due to a derivative of acetic acid, either diacetic (aceto-acetic) acid, or ethyl-diacetic acid. The interest of these facts will appear when the complications of diabetes are described.

Clinical Tests for Sugar.—The particular form of sugar contained in diabetic urine is grape sugar, or glucose ($C_6H_{12}O_6$), and this has the property, when heated with a salt of cupric oxide, of reducing it to the state of the suboxide. This test, in one or other form, is the one most commonly employed.

Trommer's Test.—To a drachm of the urine placed in a test-tube a few drops of a solution of cupric sulphate are added, and then about half a drachm of liquor potassæ, when, if sugar is present, the cupric oxide at first precipitated by the potash will be redissolved. On applying heat to the solution, a thick precipitate, at first yellow, quickly changing to orange and red-brown, is thrown down, consisting of the suboxide of copper.

The test may be applied by means of *Fehling's* solution, which consists of equal parts of (1) a cupric sulphate solution of 34·63 grammes in half a litre of distilled water, and (2) an alkaline tartrate solution of potassium hydrate 80 grammes, and sodium potassium tartrate 173 grammes in half a litre. Pavy's modification of the above is as follows:—In ten ounces of distilled water 640 grains of neutral potassium tartrate, and 1280 grains of potassa fusa; in another ten ounces, 320 grains of cupric sulphate are dissolved. The two solutions are mixed. After a time decomposition takes place, and renders the test uncertain. This may be prevented by keeping the two solutions separate, only mixing them for use, or at short intervals; or the altered mixture may be again rendered fit by the addition of a piece of caustic potash.

With Fehling's and Pavy's solutions, the test is best applied by placing 40 to 60 minims in a test-tube, heating them to the boiling point, and then adding some of the suspected urine. If heat be continued, the yellow precipitate shows itself at the upper part, and if enough urine be added, the test solution will finally lose its blue colour entirely.

Moore's Test.—Half a drachm of urine is heated in a test-tube with an equal quantity of liquor potassæ. If sugar is present the colour will gradually change to a rich red-brown, and an odour somewhat like that of treacle or burnt sugar will be given off. This is rather uncertain for small quantities, as urines free from sugar will sometimes darken with potash.

Böttger's Test.—The urine is heated with a little soda or sodium carbonate and a few grains of subnitrate of bismuth; the mixture becomes black, from the reduction of the bismuth salt to the metallic state.

Picric Acid Test.—Liquor potassæ is boiled in a test-tube with a saturated solution of picric acid. The diabetic urine is then added and the boiling is continued, when a dark claret-red colour (picramic acid) is developed, the intensity of which is proportionate to the amount of sugar (G. Johnson).

Indigo-carmine.—To a pale blue solution of indigo-carmine rendered alkaline by sodium carbonate, a drop of the suspected urine is added, and the liquid is freely boiled for a few seconds. "Then the tube should be raised an inch or two above the flame, and held without shaking, while the solution is kept quite hot, but without boiling, for exactly one minute by the watch. If glucose be present in abnormal amount, the soft rich blue will be seen first of all to darken into violet; then, according to the quantity of sugar, there will appear in succession purple, red, reddish-yellow, and finally, straw-yellow" (G. Oliver). At this stage the slightest shaking will cause red streaks to fall from the surface of the liquid, and further shaking will cause the return of the colours in inverse order up to the original blue tint. The test is conveniently carried and applied by means of the test papers devised by Oliver.

Phenyl-hydrazine Test.—About two grains of phenyl-hydrazine hydrochlorate, and three or four of sodium acetate, are added to a drachm of the urine in a test-tube; and the test-tube is kept at a boiling temperature for several minutes (20–30) by the use of a water-bath or otherwise. It is then cooled by immersion in cold water, when a yellow deposit forms, which shows, under the microscope, clusters of fine crystalline needles (phenyl-glucosazone). If the sugar is in very small quantity, the mixture should stand in a conical glass, and crystals should be sought for in the lower strata with the microscope; and it may be desirable to use the water-bath for at least an hour. But for clinical purposes the following method advocated by R. T. Williamson is much simpler, quite sufficient, and is free from the charge of excessive delicacy made against the above procedure. A test-tube is filled for about half an inch with phenyl-hydrazine hydrochlorate, and for another half-inch with sodium acetate. The test-tube is then half filled with urine, and the whole is boiled over a spirit-lamp and kept boiling for two minutes. The sediment is examined after about half-an-hour.

Fermentation Test.—If a small quantity of yeast (washed free from any starch or sugar) be added to the urine, and this be set aside in a warm place for some hours, the glucose will be converted by fermentation into alcohol and carbonic acid. If now the specific gravity be taken, and compared with what it was before the experiment, or better with that of a duplicate specimen, placed under similar conditions, except for the presence of yeast, it will be found that there is a loss of density corresponding to the destruction of the glucose. If the test-tube be filled entirely and inverted in a saucer, the carbonic acid gas, as it forms, will collect in the upper part and displace the urine.

Polariscope.—By circular polarisation not only the presence but the quantity of sugar can be accurately estimated. But the instrument is not generally available for clinical use. The glucose found in the urine rotates the polarised ray to the right, and is called *dextrose.*

Fallacies.—With the copper test a small amount of reduction may take place from the presence of uric acid, hippuric acid, and creatinine when no sugar is present. Glycuronic acid ($C_6H_{10}O_7$) reacts to Fehling's and Böttger's test, and is also dextro-rotary. It is thus readily mistaken for glucose, but it does not answer to the fermentation test; and it gives a brownish amorphous deposit with the phenyl-hydrazine test instead of the characteristic yellow needles. The supposed occurrence of glycosuria after the administration of morphine, curare, and chloroform vapour is due to this substance; chloral, butyl-chloral, and camphor also produce it.

Quantitative Estimation of Sugar.—In any cases of diabetes undergoing treatment, it is desirable to estimate from time to time, if not daily, the amount of sugar passed per diem. For this purpose all the urine passed in twenty-four hours must be collected, and a specimen of the mixed urines submitted to analysis by one of the following methods.

The Copper Test.—Pavy's solution, of which the formula has been given, is of such a strength that 100 minims are completely decolorised (from the reduction of the oxide of copper) by half a grain of grape sugar. One hundred minims should be measured into a small evaporating dish, and raised to a boiling temperature. While in this state the urine to be tested should be dropped in from a graduated pipette, and the change of colour in the blue solution should be watched till it is entirely discharged. The pipette will now show how much urine has fallen into the dish and mixed with the test; and this quantity of urine contains half a grain of sugar, since it has decolorised the cupric solution. From this the amount of sugar per ounce, and hence the daily amount, can be readily calculated.

Ammoniated Cupric Solution.—Pavy has improved on this process by using a solution containing ammonia, by which the precipitation of the suboxide is prevented, and the exact steps of the decoloration of the copper solution can be more accurately watched. The solution consists of—

Cupric sulphate . . .	4·158 grammes or		36½ grains.
Potassium sodium tartrate .	20·400	,,	178 ,,
Caustic potash . . .	20·400	,,	178 ,,
Strong ammonia (sp. gr. ·880)	300 c.c.		6 fluid oz.
Water	to 1 litre		to 1 pint.

The caustic potash and the tartrate are dissolved in some of the water, and the copper salt in another portion; these are then mixed, and when cold, the ammonia is added, and the whole is diluted to the specified bulk. Ten cubic centimetres of this solution are exactly decolorised by ·005 gramme of the sugar. As in the preceding method, a burette is used, into which the urine, previously diluted to the extent of 1 in 20, or even 1 in 40, should be placed; the nozzle of the burette passes through a cork in a small flask containing the 10 c.c. of the copper solution, diluted with 20 c.c. of water. Another glass tube in the cork allows the escape of steam. Heat is then applied to the flask, and the urine is gradually dropped in from the burette. When the colour is all discharged, the result is read off, and the desired calculation made. Pavy recommends, for clinical convenience, the use of hermetically-sealed glass tubes, each containing 10 c.c. of the ammoniated cupric solution.

Picric Acid.—As the depth of the red colour in Johnson's picric acid test (*see* p. 850) is proportionate to the amount of sugar, it may be used as a quantitative test. Johnson made a standard solution of acetate of iron, to represent the colour given by a definite strength of glucose-solution. The amount of dilution of the picramic acid required to assimilate it to the standard supplied the data for the estimation.

Fermentation.—This can be used for quantitative purposes by taking the specific gravity before and after fermentation for twenty-four hours: every degree of specific gravity lost corresponds to one grain of sugar per ounce. Thus, if the specific gravity is reduced from 1040 to 1025, there are 15 grains of sugar to every ounce of the urine. Or the gas may be collected in a graduated tube from a measured quantity of urine, as in the convenient apparatus of Gans. The graduations give the percentage of sugar.

Course and Symptoms.—The onset of diabetes is sometimes insidious; the patient only gradually notices that he drinks more fluids and passes more urine than normal; or he may complain of debility and loss of flesh rather than any alteration in his urine. In other cases the symptoms develop almost suddenly after chill, after quenching thirst with large draughts of water, after severe emotional disturbance, or after injuries.

The characteristic symptoms soon become unmistakable—namely, frequent and abundant micturition, great thirst, generally a very large appetite, physical weakness and loss of flesh. The appetite is sometimes enormous, but in other cases it is but little affected, and often fails towards the latter end. The mouth and lips are dry, the tongue large, red, raw, and "beefy"; and there is generally a sweet taste in the mouth. The teeth are carious, or become loose and fall out. The digestion is, as a rule, good, and patients may have no difficulty in disposing of the large quantities of animal food they take. The bowels are generally confined. The skin is harsh and dry, and the temperature often slightly below the normal. At the same time, nutrition is profoundly affected; the patient rapidly loses flesh, and becomes excessively weak; he is indisposed to make any mental effort, and is depressed and irritable. There is often loss of virility in men, and in women the menses may cease.

The progress of the disease is very variable; some cases begin suddenly, and end in death in the course of two to five weeks from the commencement; others may last, more or less influenced by treatment, for two, three, or four years; in other cases, again, the disease can be held in check for long periods by suitable treatment, and life may be prolonged for several years. An important point of distinction between the first and the last class of cases is that the former are progressive from the first, and are little if at all influenced by diet, since sugar continues to be excreted, even though the carbohydrates are withdrawn entirely from the food. Whereas, the latter are very favourably influenced by depriving them entirely of saccharine and starchy foods, so that not only does the sugar disappear from the urine, but the urine decreases in quantity, and all the troublesome symptoms are arrested for a time. A relapse, however, is tolerably certain to take place at some future date, to be again, or many times, controlled by diet. Eventually this fails, and phthisis, coma, or some other complication carries off the patient. The most rapid and intractable cases occur in young persons, while it is especially middle-aged and elderly patients in whom the disease is easily controlled by diet and treatment.

Cases occur in which there is persistent glycosuria, with no other symptoms of diabetes; and it is doubtful whether they should be classed with this disease. It is probable, however, that many of them ultimately become confirmed diabetics.

Complications.—In the course of diabetes a number of complications are liable to occur. The irritation of the saccharine urine may excite in women a troublesome *pruritus pudendi*, and in men, *balanitis*. The skin, besides being harsh and dry, is not infrequently the seat of *eczematous* or *lichenous* eruptions, generally dry in character. *Carbuncles* and *boils* are especially liable to occur in various parts of the body, and the former are not infrequently the cause of death. A form of *xanthoma* has also been seen in diabetes. There is sometimes *gangrene* of the toes or of an entire limb; but this is associated either with atheromatous arteries or with peripheral neuritis. The heart is not generally affected in a marked manner, but it may become weakened, and the pulse may be slow, or quick and irregular; occasionally, also, œdema of the feet, not due to nephritis, may indicate the same. Some of the most serious complications are those arising in the lungs, which become the subject of *pneumonia* or *phthisis*. The pneumonia is of the lobar or croupous variety, and sometimes results in gangrene. The phthisis is usually very rapid, bringing about death within from two to five months from its first appearance. This is a tubercular lesion, in spite of the absence of recognisable tubercles in the larynx and intestine, and even in the lungs themselves. For the course of the disease conforms closely to that of ordinary phthisis; both lungs are affected, the disease spreads from apex to base, cavities are formed, and finally, tubercle-bacilli have been demonstrated, either in the sputum, or in the lungs after death.

Vision is affected in diabetes in several ways. The most important is the formation of *cataract*, which is always symmetrical, and develops rapidly in the young and middle-aged, more slowly in old people. Other changes are:—*Defects of accommodation*, from paralysis of the ciliary muscle, sometimes coming on quite suddenly; *retinitis*, somewhat resembling that of albuminuria, with white spots and hæmorrhages; and *atrophy* of the optic nerve. *Amblyopia*, without visible ocular changes, like that of uræmia, also occurs.

Diabetic Coma.—This name has been given to a group of symptoms, chiefly referable to the nervous system, which are not infrequently the final cause of death in diabetes. The onset is often gradual and insidious, but may be indicated by loss of appetite, by a rapid fall in the quantity of urine and of sugar passed in the day, and by obstinate constipation. Sometimes there is severe abdominal pain. The patient then rather rapidly falls into a condition of collapse rather than coma. The pulse is quick and feeble, the surface cold, the features pinched, and the extremities livid. He lies with the eyes half open, taking no notice of his surroundings; and though he can be roused by a question, he answers, if at all, in a dazed manner, as if only half comprehending it. The breathing in these cases is peculiar: it is slow, deep, and sighing in character; the movements of the chest are very extensive, but without the rapidity of ordinary panting from exertion. At the same time, examination of the chest reveals nothing abnormal. The condition has been described as *air-hunger*. In many cases a sweetish, fragrant, or ethereal odour, likened to the smell of apples by some, may be noticed about the bed of the patient: it has been attributed to acetone. This condition may last from one to three days, when the pulse gets more and more feeble, the patient more apathetic, and finally quite comatose; and death, with rare exceptions, ends the scene. Occasionally there is a little muttering delirium, but rarely convulsions. In some cases the symptoms are much more rapid, without any warning; often after some excitement or unusual fatigue the patient becomes collapsed, with a quick, feeble pulse and livid extremities, and dies after twenty-four or thirty-six hours. It seems probable that there is no essential difference between these extremely rapid cases and the slower cases first mentioned, in which the peculiar character of the respiratory movements is such a prominent feature.

The nature of these symptoms has been much discussed, and various theories have been put forward to explain them. It may be noted that they may occur in almost any case of diabetes: acute cases in young people occasionally terminate thus in the course of a few weeks or months; or more chronic cases, apparently going on well, may be suddenly attacked. The rapid change from an ordinary to a diabetic diet has seemed sometimes to be the exciting cause. *Post mortem*, the lungs, liver, kidneys, indeed all the organs, may be found perfectly healthy. The blood, in a certain number of cases, presents a peculiar pink or strawberry colour, and on being placed aside a creamy layer collects on the surface, and is seen to contain a considerable quantity of fat; there is, indeed, a condition of *lipæmia*. Corresponding with this, it has been sometimes found that the capillaries of the lung are blocked by globules and masses of fat; and to this *fat embolism* the nervous symptoms have been attributed, but without sufficient grounds.

The symptoms are much better explained by the view of a blood-poisoning by some chemical substance, associated in some way with the excess of sugar in the blood. This was at one time supposed to be acetone, and the term *acetonæmia* is frequently used to describe the condition. The theory was supported by the odour of the patient, and by the reaction of the urine with ferric chloride already alluded to. But this last does not always take place in diabetic coma, it is often seen in diabetics who are not comatose, and in other diseases than diabetes—for instance, gastric ulcer, and dilatation of the stomach—and it may be quite temporary; that acetone is the chief poison is also disproved by other considerations. Stadelmann believes that the toxic agent is β-oxybutyric acid, a compound intermediate between sugar and acetone. The condition differs in many respects from uræmia.

Among other less important nervous symptoms in the course of diabetes may be mentioned—cramps; neuralgia, especially sciatic, occipital, and trigeminal; hyperæsthesia, anæsthesia, and other symptoms of *peripheral neuritis*. The knee-jerk is frequently absent, and there may be some extensor paralysis with high-stepping gait (*diabetic pseudo-tabes*). Cerebral hæmorrhage may accidentally complicate diabetes and produce coma, which must not be confounded with that due to diabetic poisoning; the presence of paralysis would distinguish the former.

A trace of albumen is occasionally found in the urine, and this is sometimes due to a tubal or interstitial nephritis.

Fatal Termination.—Over 50 per cent. of cases of pronounced diabetes die of coma, another 25 per cent. die of phthisis or pneumonia, and the remainder of Bright's disease, cerebral hæmorrhage, gangrene, carbuncle, or other complications. Among the cases dying of coma, a certain proportion are also affected with pneumonia or phthisis, from which the patients would, no doubt, have died had they not been carried off by the more rapid nervous symptoms.

Morbid Anatomy.—In many cases, especially those of short duration, the *post-mortem* appearances differ very little, if at all, from the normal: in older cases the pathological lesions due to the complications are found. The most frequent change is enlargement, with some softening and congestion of the *kidneys*, which otherwise may look perfectly normal, or may present some fatty change. Various alterations of the tubal epithelium have been noted under the microscope:—A vesicular or swollen and translucent condition of the cells of the connecting tubes by Cantani and S. Mackenzie; glycogenic degeneration of the epithelium of Henle's tubes by Ehrlich; and necrosis of the epithelium by others. The *bladder* is often hypertrophied. The *liver* presents nothing abnormal to the naked eye. It is stated to contain less glycogen than a healthy liver. The *pancreas* has frequently been found diseased. Thus it may be atrophied or converted into fat, with very little of the normal structure left, or invaded by new fibrous tissue, or in a state of suppuration; and these changes have been found in the cases of rapidly fatal diabetes, which affect young people. The *lungs*, in cases dying of coma, are generally congested and œdematous, but otherwise healthy. In other cases croupous forms of pneumonia or the acute caseating phthisis already described are found. The *heart* may present some atrophy of its muscular fibres. The *blood* is, in some cases, quite natural in appearance, in others it is black and tarry; in others, again, it is pink and creamy, as above stated. It contains from two to four times the amount of sugar found in health; and oxybutyric acid has been discovered in it. The brain, medulla oblongata, spinal cord, and sympathetic system have been often examined, and though certain changes have been described (wide perivascular spaces, thick arteries, pigment deposits outside the arteries, &c.), it does not appear that they are peculiar to, or constant in, diabetes. Peripheral neuritis occurs, but is no doubt secondary. It is with extreme rarity that any coarse lesion, such as a tumour involving the glycosuric centre in the medulla, is found as a cause of a typical case of diabetes.

Pathology.—Many views are held as to the immediate cause of the presence of sugar in the urine, and no one of them can be said to meet all the objections that can be raised against it. The view is widely held that the sugar, whether taken directly, or formed from starch in the alimentary canal, is arrested in the liver, by being converted into a less diffusible substance, glycogen, which is deposited in the hepatic cells; that this glycogen is gradually again converted, by the action of a ferment, into glucose; and that glucose is decomposed and destroyed in the blood as rapidly as it is formed, so that it never appears in any appreciable quantity amongst the urinary excreta, though it is found in the blood to the extent of ·5 per 1000. But glycogen and sugar can be made not only from the carbohydrates of the food, but also from the proteids of the food, and even of the tissues of the body. Now it may be allowed that glycosuria might arise in one of the two following ways:—(1) If the power of the liver to convert the starch or sugar into glycogen is unequal to the quantities of starch and sugar brought to it, so that the excess passes over into the hepatic vessels and general circulation. (2) If the glycogen, from whatever source, is converted into sugar with such rapidity that larger quantities of glucose are poured into the circulation than can be decomposed or destroyed in the blood. In the first case, the saccharine ingesta may be excessive (α), or the arresting powers of the liver may be deficient (β). In the second case, the quantity of glucose formed from glycogen in the liver may be excessive (γ), or the processes (whether respiratory or muscular), by which sugar is destroyed again may be enfeebled or suspended (δ).

It will be seen that there is no question of the kidneys having any share in the pathology of diabetes. They merely filter off the excess of diffusible sugar existing in the blood.

Pavy holds that the passage of sugar and carbohydrates into the general circulation is prevented or held in check by the power of the liver to fix them in the form of glycogen, and of the intestinal villi to convert them into proteids or fat; and that sugar in any quantity neither reaches the general circulation, nor is subsequently got rid of by the respiratory or other process; although a minute amount is present in normal urine and is proportionate to the quantity normally in the blood.

According to him glycosuria can only occur if the quantity of carbohydrates is in excess of the power of the liver to transform it into glycogen; and this may arise from excessive ingestion of sugar (α), or from deficient action of the liver in converting it into glycogen (β). As the first condition only occurs accidentally or by experiment, the second is for him the chief cause of diabetes.

Others think that sugar is retained in the blood from failure of the destructive processes, and Brunton explains some cases of diabetes on this view. According to Bunge, this must be the case in the usual forms of the disease; since in health the processes of decomposition are fully equal to the task of making away with much larger amounts of sugar than can proceed from the ingesta in diabetes. He suggests that such decomposition should take place in the muscles, and that the failure is to be referred to these structures.

Various explanations have been given of the way in which the nervous system influences the hepatic functions in diabetes:—By vasomotor paralysis in the liver (Pavy); by general high blood-pressure causing dilatation of the hepatic vessels (Brunton); by the influence of the nervous system upon chemical processes in muscle (Bunge); by special influence upon the formation of ferments.

But the relation of the pancreas to diabetes has also to be taken into account. Glycosuria has been produced by the complete extirpation of this gland in animals, but fails to occur if only a small portion of the gland is left *in situ*; and we have seen that in quickly fatal cases the pancreas is often diseased. The theory adopted to explain this association is that the pancreas, besides pouring the pancreatic juice into the duodenum, has an "internal secretion" (analogous to the supposed secretion of the thyroid) which influences the transformations of the carbohydrates. For instance, it is suggested that the secretion operates upon the liver, diminishing its power of forming sugar, but increasing its power of fixing and retaining it.

The temporary appearance of sugar in the urine (glycosuria) may be noticed under various conditions:—After the ingestion of very large quantities of sugar; after attacks of whooping-cough, asthma, or epilepsy; and in connection with some severe illnesses, as cholera, diphtheria, typhus or typhoid fever.

Diagnosis.—There is little likelihood of mistaking diabetes mellitus for any other definite illness; but the presence of the disease may be overlooked, and the patient treated for a vague weakness and "debility"; or the possibility of diabetes underlying one of its complications, such as carbuncles, pruritus, phthisis, or coma, may be forgotten. In all obscure conditions of weakness and emaciation, as well as in the case of carbuncles or frequent boils, spontaneous or senile gangrene, pruritus vulvæ, balanitis, impotence, double cataract or double sciatica, the urine should be tested for sugar.

Prognosis.—This has already been mentioned. It is essentially bad. Especially in young persons the disease tends to a rapid termination; while in the aged, life may be more prolonged. When the disease has lasted some time, phthisis is very likely to develop; and in all cases the risk of rapid death from coma or pneumonia must never be lost sight of.

Treatment.—The chief indications for treatment are to remove from the diet all starchy and saccharine substances, and to administer certain drugs, such as opium, which appear to have a decided influence upon the formation of sugar.

The consideration of the diet is of the first importance. Nearly all cases are benefited, and some are for the time cured, by the avoidance of carbohydrates. For this purpose one should exclude entirely bread, potatoes, sugar, fruit, pastry, and farinaceous foods; and the diet should consist mainly of meat with fat, eggs, green vegetables, tea with cream, or a very little milk. Beer and sweet wines are to be avoided, but dry sherry, sauterne, or weak brandy and water may be taken if stimulants are desired. The deprivation of bread is most seriously felt by the patients, and various substitutes have been attempted, in which the nitrogenous elements are present and the starch is, as far as possible, excluded. Thus, there are gluten bread, bran biscuits, and almond biscuits; but, unfortunately, they are all very unpalatable, and are by no means free from starch, as usually made. It is an important question how far it is wise to put a patient suddenly on a diet which need not be less abundant than in health, but which is so greatly modified; and, also, whether carbohydrates should be absolutely excluded. No doubt some patients are not improved, but rather get weaker, on a rigid diet, and for them some exceptions may be made; but the majority of cases bear very well the substitution of gluten and other special breads for ordinary bread, and are made worse if by any lapse they are allowed to take wheaten bread again. The treatment may be begun by excluding all starch and saccharine foods (potatoes, sugar, fruit), except a small quantity of bread; and then after a few days this may be exchanged for the gluten or almond substitutes.

Attention may be directed to a few special points. Fat is not harmful, and may even be beneficial; it can be taken as fat of meat, or cream, or as cod-liver oil. Green vegetables are harmless, but blanched vegetables yield sugar, such as celery, endive, and the white stalks of cabbages and lettuces. Animal soups may be taken, but must not be thickened with flour. Saccharin may be used instead of sugar to sweeten tea. The livers of animals contain glycogen, and should not be eaten; nor should oysters and other mollusca, and the interior of crabs and lobsters, for the same reason. Lævulose may be taken in quantities up to 1½ ounces daily without increasing the glycosuria.

The following list of what may and what may not be eaten or drunk will show that there is a very considerable range allowed. The patient *may eat* butchers' meat of all kinds (except liver); ham, bacon, and tongue; poultry and game; fish of all kinds, fresh, salted, or cured; real turtle, mock turtle, and ox-tail soup; beef-tea; other broths and soups not thickened; essence of beef, mutton, or chicken; Valentine's meat juice; pure meat lozenges; a very little onion, garlic, or shallot for flavouring broths, soups, or made dishes; bunch greens, spinach, watercress, mustard and cress; green lettuce picked from the white stalk; mushrooms, cucumber, vinegar; oil; pickles; eggs dressed in any way; cream cheese, cheese, butter and cream; gluten bread; almond biscuits; bran biscuits; cocoa-nut biscuits; blancmange, and custard made with cream (not milk), and without sugar; jelly, flavoured, but not sweetened; savoury jelly. Unless the very strictest dieting is required, celery and radishes may be allowed in moderation; and turnips, broccoli, cauliflower, Brussels sprouts, asparagus, seakale, vegetable marrow, and French beans may be taken if first boiled in a large quantity of water (Pavy).

He *may drink* tea, cocoa from nibs; Liebig's extract of meat; chocolate without sugar; cream; soda or potass water; Vichy, Vals, Royat, German seltzer, or Apollinaris water; dry sherry, claret, dry sauterne, Burgundy, Chablis, hock; brandy or old whisky.

He should *not eat* sugar or starch in any form; ordinary, brown, whole meal, or aërated bread, either plain or toasted; rice, arrowroot, sago, tapioca, ordinary macaroni, or vermicelli, semolina, or any other farinaceous preparation; potatoes, carrots, parsnips, tomatoes, peas, broad beans, scarlet runners, cabbage, artichokes, endive, beetroot; pastry and puddings of any kind except those stated above; fruit of any kind, fresh or preserved; preserved ginger; oysters, or the interior of crabs, lobsters, &c.; any kind of liver; milk, except in small quantity.

He should *not drink* beer of any sort; sweet or sparkling wines or liqueurs; lemonade or any sweetened aërated drinks.

Certain drugs have an undoubted influence in controlling the excretion of sugar in diabetes, but it is always desirable at the same time to restrict the diet, at any rate until the urine is free from sugar, when sometimes the diet may be gradually relaxed, and the drug treatment maintained with a satisfactory result. The drug that has the most decided influence is opium; it should be given in doses of ½ to 1 grain twice or three times a day, and gradually increased until, if necessary, it reaches 4, 5, or 6 grains in the twenty-four hours. Under such treatment, the sugar may entirely disappear, and the patient regain flesh or strength; and if this condition is maintained, the effect of returning to a normal diet, and gradually diminishing the daily dose of opium may be tried, sometimes with success—too often, however, with the result that all the symptoms reappear. In any case the opium should only gradually be reduced to smaller and smaller doses, and should not be suddenly withdrawn.

The alkaloids of opium—morphia and codeine—may be separately employed. Pavy has strongly recommended codeine, which is given at first in ½-grain doses three times daily, and may be increased to a daily administration of 20 or 30 grains. Morphia appears to have been more successful in some cases recorded by Mitchell Bruce; a daily dose of 5 or 6 grains may be gradually reached, if required. Bruce found that the effect upon the excretion of sugar was greater when morphia was taken by the stomach, than when it was injected subcutaneously. On the other hand, its influence on the sensorium was greater in the latter case. Small doses of nux vomica will often neutralise the constipating effects of opium and its alkaloids.

The alkaline carbonates seem to be beneficial, and many patients are sent to drink the natural waters of Carlsbad, Vichy, or Neuenahr. Other remedies have been vaunted from time to time—*e.g.*, carbolic acid, salicylate of sodium, jambul seeds, and uranium nitrate. German writers advocate Clemens's solution of arsenite of bromine in doses of 3 to 5 minims after meals.

Of the complications, pruritus and eczema will probably improve as the sugar is diminished; borax ointment is a good application for the former. The treatment of diabetic coma has hitherto been almost hopeless. Fagge, myself, and others have injected into the veins a solution of phosphate and chloride of sodium, following the idea that the blood might be unduly viscid; but without any good result. Stadelmann, in accordance with his theory that the symptoms are due to oxybutyric acid in the blood, recommends the injection of an alkaline solution containing bicarbonate of sodium. As a rule, one has to be content with administering diffusible stimulants, such as brandy, ammonia, ether, or camphor, placing hot bottles to the feet and legs, or placing the patient in a hot bath. The preceding constipation tempts one to use purgatives, but they are often entirely inoperative.

FUNCTIONAL ALBUMINURIA.

It has been already shown that albuminuria may occur in a number of morbid conditions, of which nephritis and renal degenerations, acute illnesses, infectious diseases, and venous congestion are the most important. Indeed, in the majority of cases in which albuminuria is found, the cause can be referred to one of these groups. But it is occasionally present in persons who appear to be in perfect health, or, at any rate, in working health. It may occur without any apparent cause, or it may be brought on by an exercise of function which in other individuals is quite harmless —*e.g.*, a full meal, especially of albuminous food, muscular effort, or exposure to cold. It may be of short duration, quickly passing away to recur from time to time, or it may persist for months.

Different cases of this disturbance have been described under the names, physiological albuminuria, albuminuria in the apparently healthy, intermittent albuminuria, remittent albuminuria, cyclic albuminuria, postural albuminuria, and functional albuminuria.

The first of these names is clearly inapplicable. It is true that Leube found albumen in five out of 119 soldiers in the morning, and in nineteen out of the same number of soldiers after exercise; and that Fürbringer found albuminuria in seven out of eighty-one children in the afternoon. Stirling also found albuminuria in 25 per cent. of 461 individuals, including boys on a training ship, ships' officers, paupers, cement workers, and brewers' men. But there are many slight lesions, not interfering with practical working health, which nevertheless are not normal nor physiological. The term functional albuminuria has, perhaps, too wide an application, as it might fairly include albuminuria in heart or lung disease, where the kidney, at least in early stages, is as yet uninjured. Other terms describe the conditions as to time under which the albumen appears, with the implication, not always true, that in actual disease of the kidney albuminuria is persistent.

The following are different forms of functional albuminuria that have been described :—

1. *From errors of diet* (*dietetic albuminuria*).—The chief cause is the ingestion of large quantities of albuminous food, such as eggs; in some persons any excess of food. Albumen is occasionally associated in the urine with crystals of calcium oxalate, which may possibly irritate the kidneys, and thus cause the passage of albumen; and such excess of oxalates may certainly be produced by particular kinds of diet (*see* p. 775). On the other hand, in some cases of transient albuminuria with oxalates (Moxon's *intermittent albuminuria*), no special diet seems to have been indulged in, and the patients were often slightly out of health—that is, languid and weak, with deficient appetite, conditions recognisable less by the patient than by his friends.

2. *From exercise.*—A temporary albuminuria has been known to occur after running a long distance, and after other severe muscular efforts.

3. Possibly related to the last is the group of cases which Pavy has called *cyclic albuminuria.* It occurs in persons in good health, between the ages of nine and forty-nine, and is characterised by the presence of albumen at certain times in the day, while it is absent at others. Thus, in the early morning it cannot be detected; it is present from about 9 A.M. to 5 or 6 P.M., and again disappears from the urine passed at night. The albumen has been shown to depend entirely upon the assumption of the erect position (*postural albuminuria*), and accompanying exercise during the day; and it disappears as a result of the recumbent position at night. The amount of food taken has no influence upon it. Pavy observed that pure serum-albumen was not present in these cases, but a mixture of serum-albumen with alkali-albumen, precipitable by the organic acids, such as citric. Moxon alluded to what must have been similar instances under the term *remittent albuminuria.*

4. *Disturbed innervation* may be the cause of transitory albuminuria, *e.g.*, mental anxiety and prolonged study; and perhaps in this way may arise cases which are due to masturbation.

5. *Paroxysmal albuminuria.*—This was regarded by Fagge as a mild form of paroxysmal hæmoglobinuria, in which the hæmoglobin broke up into hæmatin and globulin; and Ralfe held a similar view, suggesting that it is due to an increased hæmolytic action of the liver. The patients have malaise, a sallow tint, and subsequently albuminuria; the urine contains at the same time excess of urea and of urobilin.

6. Cases of albuminuria persistent for years, without any other

signs of Bright's disease, are described; but they must be admitted with great caution into the present category.

The explanations of these varieties of albuminuria are still much discussed; and they are likely to vary with the views held as to the pathology of albuminuria in general. Abnormal diffusibility of albumen, increased pressure in the glomeruli (as from exercise or the erect posture), and vasomotor paralysis from modified innervation, are relied upon to explain some cases.

Diagnosis.—In order to distinguish these cases from the graver instances of Bright's disease, a single examination of the urine is seldom sufficient. If albuminuria is discovered, as it often is, in the routine examination of the urine for life insurance, or admission to a school, a city office, or to the public services, where the individuals are presumably healthy; or if it is found in a young person, who is at most only a little languid and out of sorts, a diagnosis of Bright's disease should not be hastily made, but the urine passed at different periods of the day should be tested to see if the albuminuria is determined by any of the factors—diet and position—alluded to; and especially to see if it is at any time absent. In chronic Bright's disease, the albumen fluctuates during the day, but the entire absence at night and during the early morning is very characteristic of the functional forms. The albumen is always small in quantity; casts are, as a rule, not found, except in the cases brought on by exercise, but they should be sought by the aid of the centrifuge; the pulse shows no high tension, and the heart is normal.

Prognosis.—The immediate future of these cases is on the whole favourable, and in the large majority cure takes place; but the occurrence of such a disturbance of the renal function suggests that either the kidneys themselves or the tissues of the body generally may not have quite their normal powers of resistance.

Treatment.—Where the cause can be recognised, such as diet or exercise, this should be altered or avoided. Where calcium oxalate seems to be responsible, dilute nitro-hydrochloric acid may be given, and meat, excess of vegetables, and wines should be excluded from the diet. In cases of cyclic albuminuria, where the health is maintained, no treatment seems necessary; the urine should be watched from time to time, and any other symptoms that arise must be met by suitable remedies.

CHRONIC INTOXICATIONS AND THE EFFECTS OF HEAT.

ALCOHOLISM.

UNDER this term are included the results of the more or less continued use of alcohol, not the immediate effects of a considerable overdose, known as drunkenness or intoxication. The symptoms of the latter are familiar, and usually subside as the alcohol passes through the system. Occasionally, however, from the rapid imbibition of large quantities of spirit in those unaccustomed to it, a fatal result may ensue. It is preceded by unconsciousness, with pupils sluggish or fixed, dilated, or contracted; small pulse, cold, clammy skin, stertorous respiration; and sometimes delirium or convulsions.

DELIRIUM TREMENS.

This commonly occurs in those who habitually drink freely, who may have been not infrequently drunk, and who have recently been taking unusual quantities continuously for some days. It is often indeed stated that the patient had left off drinking for two or three days before the symptoms came on; but as a distaste for drink is sometimes one of the first manifestations of the disease, it is probable that this is an explanation of the apparent anomaly. Delirium tremens is also sometimes determined in habitual drinkers by some severe shock, such as the fracture of a bone, or the onset of pneumonia, erysipelas, or other acute disease, without evidence of any amount of drinking beyond the daily average.

Symptoms.—The first symptoms are disturbed sleep, restlessness, irritability, and loss of appetite; and with the disturbed sleep there are unpleasant dreams, and some wandering. In the morning the patient may be more rational, but the delirium returns at night; the next day the delirium continues, and shows its characteristic features. The patient is constantly talking, addressing either those about him, or imaginary persons whom he supposes to be present. He talks on his business, or on other affairs, frequently changing from one subject to another. He may be recalled to his senses for a moment, but soon relapses. In the pursuance of his thoughts he may try to get out of bed, or pull the bedclothes about. With this there is very marked tremor: the hands shake with quick oscillation whenever he moves them: the lips and tongue tremble when he speaks. As the condition gets worse he has very definite hallucinations and illusions. Objects in the room or figures on the wall-paper are conceived to be animals or insects: he is constantly seeing cats, dogs, rats, mice, or blackbeetles running after him or crawling about the bed: he looks under the bed, or behind the curtains, or peers behind any bystander, and is suspicious of injury from those about him. He may, under some such false impression, strike those who are near: but, as a rule, he can, with a little firmness, be controlled, and it is often easy to turn his thoughts for a moment in another direction.

Other symptoms accompany this mental and muscular disturbance. The face is flushed, the conjunctivæ are suffused, the tongue is thickly furred and becomes dry, as the disease progresses. The pulse is quick, soft, at first full, afterwards small and feeble: the temperature rises to 102° or 103°, and there is occasionally hyperpyrexia (106° or more). The skin is generally moist or even profusely perspiring: and the urine is accordingly dark-coloured, scanty, and of high specific gravity. It may contain a small quantity of albumen. According to Gowers there may be indications of early optic neuritis.

About the end of the third day, with considerable constancy in favourable cases, some improvement begins to show itself. Hitherto sleep has been entirely absent, but now he falls into a slumber which may last eight or ten hours, and he wakes much refreshed, the delirium and trembling are less, and gradually improvement takes place in all respects. In more serious cases the face loses its colour and becomes pale and earthy, the pulse is quick and feeble, the delirium is less active, the patient more prostrate: he lies on his back muttering, and semi-comatose. Finally death takes place, preceded, it may be, by convulsions or hyperpyrexia. In some instances the symptoms are comparatively mild, and subside in a day or two: in others, the long-looked-for sleep is not directly curative, but is repeated more than once before recovery is ensured.

Death occurs from exhaustion: from cardiac failure, and this is sometimes sudden: or from pneumonia.

Morbid Anatomy.—The changes are not very pronounced: some congestion of the cortex of the brain, of the bases of the lungs and of the kidneys, may be all that can be found. Thickening and opacity of the membranes of the brain is a chronic condition not responsible for the acute symptoms.

Diagnosis.—It may be simulated by *meningitis*, and apparently by *general paralysis of the insane*. The former is generally distinguished by early convulsions, or later on by paralysis, sometimes, but not always, by headache, or by the duration of the symptoms. In the latter, a carefully studied history ought to help to a right conclusion. In all cases of delirium tremens it should be remembered that pneumonia or fever may be present.

Treatment.—The patient should be kept as quiet as possible in a darkened room undisturbed by visitors. If he is restless and getting out of bed, male attendants are desirable who may with a little management keep him under control. Extreme violence may require a strait-waistcoat, or a sheet stretched over the trunk, legs, and arms, and fixed to the sides of the bed; or broad padded leather bands, by which the shoulders, wrists, and ankles can be fixed down to the bedstead, thus leaving the chest free from restraint. These methods should, however, if possible, be avoided. The likelihood of the patient doing injury by getting at knives or forks, by drinking lotions or liniments, or by jumping out of the window must be borne in mind. Food must be given every two or three hours in small quantities; it may be milk and beef-tea, and it is better if it can be peptonised by the use of liquor pancreaticus or similar preparations. The use of drugs has to be approached with great caution. Narcotics seem to be called for, and bromide of potassium, chloral, opium, morphia, and sulphonal are those which may be employed. But there is, in delirium tremens, a remarkable resistance to the action of such drugs, and the fear is that, finding the usual doses are inoperative, one may proceed to larger and larger quantities, which remain in the body, and are only too dangerously effective as the disease is subsiding. Fifteen or twenty grains of chloral may be given every six hours; or a quarter of a grain of morphia or $\frac{1}{150}$ grain of hydriodate of hyoscine may be injected, and repeated at the same interval, if required. Stimulants are better avoided, unless they become absolutely necessary in the stage of prostration, when ammonia, ether, or alcohol may be given.

CHRONIC ALCOHOLISM.

The effects of chronic alcoholism are seen most markedly in various nervous symptoms, in impaired digestion, and in cirrhosis of the liver; it no doubt also contributes to fatty and to atheromatous degeneration of the arteries, and to disease of the kidneys. These different parts of the body may be affected separately or together; but, as a fact, the term chronic alcoholism is more commonly applied to those cases in which the nervous system is involved, and less often to those in which the liver alone, or the kidneys alone, are the seat of disease. In all cases it is important to observe that it is not drunkenness or intoxication which leads to this condition, but the constant imbibition of considerable quantities of beer, wine, or spirits, possibly without the individual getting drunk on any single occasion. The daily amount of alcohol and the duration of the drinking habits required to produce this effect in different persons are very variable.

Symptoms.—*Nervous System.*—Muscular tremor is one of the first indications. The hands are unsteady, and the tongue trembles when it is protruded. The patient is restless and irritable, sleep is bad, and he wakes unrefreshed, with a feeling of prostration that tempts him to take stimulants at once. Sinking feelings are frequently complained of, which require, according to the patient, to be met by fresh doses of the favourite drink. As things get worse, the patient is unequal to any sustained mental effort; even the simplest business transaction must be preceded by a glass. Sensory disturbances may be also present, such as buzzing or rushing in the head, vertigo, muscæ volitantes, flashes of light, or diffused headache. There may be severe neuralgic pains in the legs, possibly the early signs of neuritis. In later stages the mind is seriously involved. Judgment, intellectual capacity, volition, and the moral sense are all weakened. The patient becomes hesitating and vacillating, unable to follow out any definite line of action, but unscrupulous in his attempts to get stimulants at all times. Multiple neuritis (*see* p. 179), epileptic fits, irregular paralysis or anæsthesia, and some forms of insanity, which may be mania, melancholia, or dementia, also occasionally result from continued excess.

Gastric Disturbance.—This is generally shown quite early, coincidently with the first nervous symptoms. The patient vomits in the morning directly he rises from bed, he is quite unable to eat any breakfast, and his appetite generally is deficient. The tongue is covered with thick yellowish fur, and the breath is fœtid. The eyes are suffused, and the face may be tinged with yellow.

In course of time, the minute venules of the cheeks become dilated, the nose is red and thickened, and sometimes acne rosacea develops. The face becomes more and more bloated, and the blotching with dilated venules is more marked. In this stage, or a little earlier, the liver may be found to project 2 or 3 inches below the costal margin, and the urine frequently contains albumen. The occurrence of dilatation of the heart under these circumstances has been referred to (*see* p. 545).

The early tendency to obesity gives way as things get worse to the converse condition, and it is not uncommon to see the patient with wasted chest, arms, and legs, and the abdomen large from retained fat, or cirrhosed liver and ascites; the feet are perhaps œdematous, while the urine is scanty (*see* Cirrhosis of the Liver, Chronic Interstitial Nephritis, and Gout).

In addition to the diseases directly resulting from alcohol, these patients are liable to succumb with great rapidity to any acute illness, such as pneumonia, erysipelas, or typhus.

Pathology.—In chronic alcoholism the membranes of the brain are commonly opaque and thickened, and the convolutions are shrunken and atrophied. The changes in neuritis, hepatic cirrhosis, &c., are described elsewhere.

Treatment.—The one essential is the abstention from alcohol in any form, and it is desirable that this should be immediately enforced, with no attempt at diminishing the quantity day by day. The patient will advance all sorts of excuses as to why he should not give it up at once, but they should not be regarded. No drug is of any value as long as the drinking is continued, but the craving may be perhaps diminished, and the patient generally benefited, by tonics, such as quinine, cinchona, nux vomica, and cod-liver oil. To obtain sleep, bromide of potassium, chloralamide, trional, paraldehyde, hyoscyamus, Indian hemp, and rarely morphia may be employed. Where patients will submit to the restraint, the *régime* of a hydropathic establishment is eminently suitable.

LEAD-POISONING.

(*Plumbism.*)

This occurs among those who work in lead, or lead salts, such as painters, plumbers, type-founders, and compositors, and accidentally as the result of impregnation of drinking water with lead from the cisterns in which it is stored, from taking snuff that has been packed in lead paper, from the use of hair-dyes, or otherwise. It may thus enter by the alimentary canal—the most frequent way—by the skin, or occasionally by the respiratory mucous membrane. Like alcohol and other poisons, it acts very differently on different individuals; some men are attacked with characteristic symptoms within a few months of entering a white-lead factory; others may work in it for years with impunity. The tendency to be attacked is increased by starvation, ill-health, exposure to cold, and indulgence in drink (T. Oliver).

Symptoms.—The effects which it produces on its victims are—(1) colic; (2) paralysis and other nervous symptoms; (3) "blue line" on the gums; (4) anæmia; (5) a tendency to the occurrence of gout or granular kidney; (6) disturbances of the generative organs.

1. *Lead Colic.*—This is a form of intestinal colic. The patient is seized with severe spasmodic pain at or around the umbilicus;

the abdominal muscles are contracted, and the pain is rather relieved by firm pressure. Sometimes there is vomiting and the bowels are nearly always confined. The pain diminishes for a time and then recurs; and in the intervals of the spasmodic attacks the abdomen may be tender. It is generally relieved in the course of one to three or four days. It is probably due to spasmodic contraction of the intestine, with some diminution of secretion from the intestinal mucous membrane; but exactly how these are caused still remains obscure. Oliver notes that during colic the pulse is slow and hard, and the urine scanty. *Vomiting* is sometimes a troublesome symptom before the occurrence of colic.

2. *Lead Paralysis.*—The characteristic feature of lead paralysis is the affection of the extensors of the hands, and the consequent "dropped wrist." If the arms are held out with the forearms pronated, the hands hang down, and the patient is unable either to raise them or to extend the fingers. If the hand and the first phalanges are supported in the horizontal position, the remaining phalanges can be extended, showing that the lumbricales and interossei are still active. Indeed, the paralysis may be and is often confined to the extensors of the fingers, the lower two extensors of the thumb, and the extensors of the wrist. The extensor ossis metacarpi pollicis and the supinator longus generally escape. The failure of extension is most marked in the little finger, least in the forefinger. After a time the muscles of the back of the arm waste, and a peculiar prominence forms on the back of the wrist, due to a backward displacement of the bones of the carpus, and possibly to a distension of their synovial sacs. Examination with the battery shows reaction of degeneration; faradism applied to nerve or muscle gives no result, and if applied to the extensor muscles it commonly acts through these and causes contraction of the flexors. With the galvanic current there is increased contraction of the muscles, and greater activity with the anode on the muscle (ACC), than with the kathode (KCC).

Sometimes other muscles of the arm are affected, especially the deltoid, the biceps, the brachialis anticus, and the supinator longus; in the legs, the long extensor of the toes and the peronei. Quite rarely there is weakness without wasting in the upper arms or thighs, or a universal loss of power. The interossei and small muscles of the thumb and little finger are also paralysed in lead-poisoning not infrequently, but, according to Gowers, they are more frequently affected with a form in which wasting and weakness come on simultaneously (primary atrophic), than with the above described lesion where weakness comes on first (degenerative). In this primary atrophic form the reactions to the galvanic and faradic currents are in proportion to the degrees of wasting, as is the case in progressive muscular atrophy. The two varieties may occur together.

Sensory symptoms are rarely present with lead paralysis; there may be darting pains, slight anæsthesia, and occasionally tremor.

Independent of paralysis, chronic lead-poisoning may cause dull aching pains in the muscles or joints, often with tenderness in the muscles, and tingling and irregular anæsthesia in the limbs. These are not unlike the sensory symptoms of alcoholic paralysis, and may be due to neuritis.

Cerebral Symptoms.—Saturnine Encephalopathy.—These may be in the form of hemiplegia, or of hemianæsthesia. More severe cases, not infrequently fatal, occur in which convulsions, delirium, and coma, with, perhaps, optic neuritis and some fever, are the symptoms. Such cases often run a very acute course, and appear to be more frequent in females (Oliver). Anæmia is the first symptom, and then colic, headache, vomiting, diplopia, or defective vision from optic neuritis. In a few days the patient is convulsed, becomes comatose, and dies. In other instances, there is mental disturbance, amounting to insanity, which is either acute mania or melancholia, or progressive mental failure and muscular weakness, with, perhaps, convulsions.

Ocular lesions and symptoms are frequent, and include optic neuritis with or without hæmorrhages, neuro-retinitis, primary and consecutive atrophy, inequality of pupils, diplopia, and a bilateral amblyopia without change in the fundus, similar to uræmic amaurosis.

3. The *blue line* on the gums, or *lead line*, has mainly a diagnostic importance. It shows that lead has been taken into the system. It is a dark slate-coloured or black finely-dotted line, which forms in the gum close to the teeth, and consists of a deposit of sulphide of lead in the tissues around the vessels; this results from the union of lead with sulphur provided by albuminous substance (partly contained in "tartar") at the edge of the gum. Where teeth are absent there is no blue line, and if the teeth are kept exceptionally clean, or the gum lies close up to the teeth, the blue line is absent. In such cases, it may be seen only in portions of gum rising between the teeth. It may exist without any other symptom of plumbism; it persists for from eight days to three months (Oliver), or much longer, according to some others, after all further entry of lead into the system has ceased.

4. Sufferers from lead-poisoning are generally *anæmic*, often remarkably so, with a sallow earthy look. The red corpuscles are diminished in number, and contain less than their normal amount of hæmoglobin. The anæmia is often the earliest indication of impregnation with lead.

5. The relation between lead, *gout* and *granular kidney* is very intimate. Chronic lead-poisoning leads to a diminished excretion of uric acid, and gout is frequent in those who suffer from lead-poisoning. Conversely, it has been stated by Sir A. Garrod, that persons who have suffered from gout are readily affected by lead given medicinally. Albuminuria is often found to occur in chronic lead-poisoning, and sometimes this is certainly due to the kidneys becoming granular.

6. The disturbances of the *generative organs* which occur are abortion in pregnant women, and menorrhagia in others.

Pathology.—Lead has been found in various organs, namely, the brain, liver, and spleen, also in the bones; but in very small quantity. It obviously interferes in some way with the formation of blood, as shown by the anæmia, and profoundly modifies other metabolic processes in the body, as seen by its relation to uric acid. The cæcum and colon have been found blackened by sulphide of lead. The paralytic symptoms are mainly due to a neuritis, which has been found most marked in the intra-muscular twigs; it is less so in the larger trunks, and is usually absent in the parts near the nerve-roots. In a few cases atrophy of the nerve-roots has been seen, and in some even changes in the anterior cornua, but, as a rule, the spinal cord is quite healthy. In the muscles the usual degenerative changes are found (p. 177).

Diagnosis.—This depends for the most part upon the history of lead-poisoning, upon the presence of a lead line upon the gums, and upon the discovery of lead in the urine. The history may have to be investigated with the greatest care, as lead may get into the system by the most unexpected means. If paralytic symptoms are present, a history of colic is often obtainable, and in nearly all cases the lead line is present, though in very cleanly persons it may only be found between the teeth, or possibly not at all. Simple double paralysis of the extensors of the forearms is probably due to lead; but if the small hand muscles are involved, the resemblance to *progressive muscular atrophy* is very close. The urine should be examined after iodide of potassium has been taken for a week, as the effect of this drug is to cause lead deposited in the system to be taken up by the circulation and secreted by the urine.

Prognosis.—Typical wrist-drop often recovers, but it may be very slowly. Severe cerebral symptoms endanger life, but if death does not occur, recovery may be complete.

Treatment.—The patient should give up his occupation, or prevent in whatever way may be necessary the introduction of more lead into the system.

Colic generally yields readily to a full dose of opium combined with a simple purgative; an ounce of castor-oil with 15 or 20 minims of tincture of opium is a common and successful prescription. Sometimes it may have to be repeated; a strong saline purge may be given, or a drop of croton oil may be added to the castor oil. Hypodermic injection of morphia may be used instead of opium. Hot fomentations or poultices should in the meanwhile be applied to the abdomen. Iodide of potassium in 5-grain doses, three times daily, should be given for two or three weeks afterwards.

In *paralytic affections* the iodide should also be given, and the muscles galvanised with the continuous current; if after some time they react well to the faradic current, this may then be substituted. Massage may also be tried. For *acute cerebral attacks* Oliver recommends the inhalation of nitrite of amyl.

Anæmia must be met by suitable ferruginous treatment in addition to what is necessary to prevent further impregnation, such as removal from work and the use of potassium iodide.

Prevention.—It is unnecessary to say much on this point. The protection of lead-workers is a matter for the employers of labour, or, failing them, for the State. It is obvious that cisterns or pipes used for drinking-water should not be constructed of lead, nor should recourse be had to this metal for the preservation of anything, such as food, drink, snuff, or tobacco, which is to be afterwards taken into the system.

CHRONIC MERCURIAL POISONING.

This is chiefly brought about by the inhalation of the vapour of mercury in those who work in quicksilver mines, and in making mirrors, barometers, and thermometers; it may or may not be associated with the salivation and stomatitis which are the familiar results of overdosing with mercury used as a drug.

The characteristic of this form of poisoning is the tremulous movements of the limbs and body known as "mercurial tremors," or the "trembles." They come on suddenly or gradually, generally after some years' exposure to the metallic vapours. They affect the arms first, and then spread to the legs and the muscles of the rest of the body. The movements are at first brought out only by excitement, later whenever the patient makes any muscular effort, and finally they become constant, and even persist to a certain extent during sleep. The tongue is tremulous, and speech is hurried, abrupt, and jerky. The patient may be quite unable to walk without support. In the earlier stages there is some resemblance to disseminated sclerosis, but there is no nystagmus; and if the movements become constant, paralysis agitans is, to a certain extent, simulated. Various cerebral symptoms, such as delirium, hallucinations, and mania, occur in extreme cases.

Recovery may take place if the disease is not too advanced.

Treatment.—Removal from the fumes of mercury is essential. Tonics and iodide of potassium are of most value as drugs.

INSOLATION.

(*Sun-stroke. Heat Apoplexy. Thermic Fever.*)

Insolation occurs only occasionally in the hottest summer in the British Isles, but frequently in India and other tropical parts. It has been generally regarded as the result of heat upon the body, though not necessarily caused by the direct action of the sun's rays. Thus the symptoms develop in barracks, workshops, and similar buildings, almost as often as in the open air. A moist atmosphere is more favourable than dry air, and the individual is predisposed to it by habits of intemperance in general, and by excessive exertion at the time.

Symptoms.—The symptoms are not always the same, and it has been usual to describe three forms: a syncopal, an asphyxial, and a hyperpyrexial form. The view has recently been advanced by Sambon, with whom Manson agrees, that the hyperpyrexial form is really an infectious or even epidemic disease occurring, it is true, in hot countries, but not determined solely by the effects of the sun's rays in a hot atmosphere upon the bodily tissues. According to this view the syncopal form is merely a cardiac failure, which may occur in any country should the heat become extreme, whereas the hyperpyrexial form, or *Siriasis*, is limited to the tropical or subtropical countries in which it is endemic.

Syncopal or Cardiac Form.—The patient suffers from faintness, or complete syncope, nausea, or vomiting; the surface of the body is pale, cool, and moist, and the temperature is subnormal; the pulse is quick and feeble; the pupils are dilated. Death may take place from continued heart-failure, but recovery is frequent.

Asphyxial Form.—In this the symptoms come on quite suddenly—a true stroke. The same collapse and cardiac failure are present as before, but respiration fails as well.

Hyperpyrexial Form or *Siriasis.*—This often develops gradually, the early indications being weakness, restlessness, and sleeplessness, nausea or vomiting, thirst and anorexia, giddiness or headache, hurried breathing or præcordial anxiety, and often profuse and frequent micturition. There may be incoherent talking, or a temporary state of delusion; the patient becomes unconscious, with laboured stertorous breathing, rapid feeble pulse, contracted pupils, and livid or congested face; and the temperature is found to reach 109°, 110°, or 111°. This may be succeeded by convulsions, suppression of urine, and death.

Anatomical Changes.—Very little is noted in the cases of rapid death from cardiac or respiratory failure. In the hyperpyrexial variety the blood remains fluid, the heart is contracted from coagulation of its myosin, the lungs are intensely engorged, with hæmorrhage under the pleura, the cerebral meninges are congested, and it is said that there may be evidence of early meningitis.

Prognosis.—The mortality is stated to be from 45 to 50 per cent., and in cases that recover sequelæ are often observed, which may be, according to Fayrer, weakness from obscure structural change in the cerebrum, or chronic meningitis, epilepsy, defective memory, nervous irritability, headache, insanity, partial paraplegia, partial or complete blindness, or extreme intolerance of heat. The syncopal form is least likely to be attended with sequelæ.

Treatment.—Removal from the source of heat and the use of the cold douche are necessary in all varieties. In the syncopal form the douche must not be too long continued, and the condition of the pulse may require direct stimulation. In the hyperpyrexial form the temperature must be reduced as quickly as possible; if ice can be obtained it should be put in the water used, or it may be rubbed directly over the body of the sufferer, until the temperature in the rectum has fallen to 104°. After this it will fall to the normal unassisted. Manson objects to chemical antipyretics, but recommends the subcutaneous injection of 40 minims of tincture of digitalis. For sequelæ, removal to a cool climate, and the use of iodide of potassium and counter-irritants are advised.

DISEASES OF THE BONES AND JOINTS.

ACUTE RHEUMATISM.

THE terms rheumatism and rheumatic correspond in their wider acceptation to the ideas of pain, of an origin in cold or damp, and of an affection of the joints. Thus, a typical rheumatism would be a painful joint affection resulting from cold or damp. In its etymology, the word rheumatism—from ῥέω, I flow—is connected with catarrh, and hence with the idea of cold. The loose way in which it has even recently been employed in medicine is shown by such names as "rheumatic facial paralysis," meaning facial paralysis from exposure to *cold;* "syphilitic rheumatism," in which neither joints nor cold are in question, but only a *painful* periostitis; "rheumatic gout," which, if it signifies anything beyond gout, must mean a gout in which many *joints* are affected. But, in the present day, acute rheumatism or rheumatic fever is taken as the type of the disease, and we should, as far as possible, limit our use of the term rheumatic to such lesions as can be shown to be directly associated with it. Acute rheumatism is characterised by inflammation of several joints at the same time or successively, with pyrexia, profuse sweating, and a tendency to inflammation of the endocardium, pericardium, or pleura.

Ætiology.—The disease occurs in both sexes, and at nearly every age; but it is very rare after fifty years of age, and in infants. It occurs with greatest frequency in its most perfect form in adults between fifteen and thirty, and quite commonly, though in a less typical manner, in young children. The tendency to its occurrence is, to a certain extent, hereditary. Its most constant exciting causes are cold and damp, and these are found to be in operation in a large proportion of the cases. It is, no doubt, in accordance with this that it is least frequent in England in the months of July, August, and September, and that the sufferers from rheumatism include a large proportion of servant girls and others of the poorer classes. Rheumatism has an important relation to chorea, which has been mentioned in the chapter on the latter; and scarlatina may be followed by a multiple synovitis, which, if sometimes certainly septicæmic, is at others quite indistinguishable from acute rheumatism.

Symptoms.—*Arthritis.*—The onset of acute rheumatism is sometimes quite sudden, so that the patient first feels a pain in one joint, and then successively in others; or there are a few days of obscure illness before the pains in the joints occur to mark its nature. There may be a little sense of chilliness, but there is rarely a distinct rigor such as occurs in pneumonia or pleurisy. The knee is often first attacked, and then the ankle; in other cases the wrist or the shoulder. Whichever is first attacked, the disease may soon spread to other joints of the body, so that the shoulder, elbow, wrist, and phalangeal joints, the hip, knee, ankle, and phalangeal joints of the toes, may all be inflamed at the same time or successively. Not infrequently the sterno-clavicular joint, and even the vertebral and costo-vertebral joints, are undoubtedly affected. But the extent of the disease is very variable. In one only two or three joints may be inflamed; in another a great number; and an important feature of acute rheumatism is the way in which some inflamed joints will quickly recover, while others become involved; and these last will get well, while fresh joints suffer, or those first affected become again inflamed. The disease flies from joint to joint.

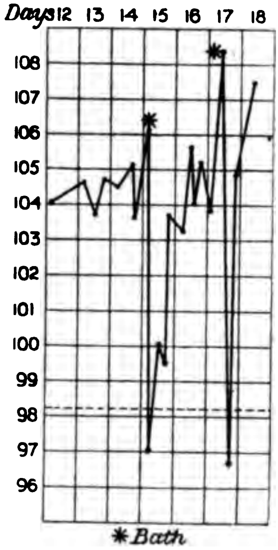
A joint attacked by acute rheumatism is swollen, red, hot, tender to touch, and painful. The swelling is most manifest in the knee, where effusion can easily be recognised, in the ankle, in the wrist, and in the joints of the fingers. The colour is mostly a bright pink, and not the dark red of gout and some erythemata; it rarely covers the whole swelling, and may be in patches. The tenderness is sometimes extreme, so that a slight shock on the bed, and any clumsy handling of the joint, will cause intense pain. It may persist after spontaneous pain has subsided. In the shoulder, hip, and elbow joints, pain and tenderness are the chief evidences of rheumatism, as slight swelling is not easily recognised, and redness is generally absent. The joints have been found in fatal cases to contain a turbid synovia, with shreds of fibrin. Leucocytes are present, but the fluid is never purulent. The synovial membrane itself is vascular, and covered with a layer of lymph. Probably the joint-changes are even slighter than this when such rapid subsidence takes place as is often witnessed.

It has been stated that the synchondroses are also sometimes involved in rheumatism. Undoubtedly the sheaths of the tendons about certain joints are often inflamed, especially those about the wrists and ankle; and some of the redness that extends on to the dorsum of the foot and hand may be due to their inflammation.

excessively high temperature, and that visible lesions of the brain or its membranes are absent. Hyperpyrexia is not restricted to any particular class of case; it may supervene equally in one that appears well on the road to recovery, and in one that has threatening lesions of the chest. It is not determined by any considerations of age, sex, occupation, previous illnesses, climate, or season; but it seems to occur with greater frequency in some years than in others.

In a certain proportion of cases, some warning is given: the joint-pains subside rather suddenly, and the sweating ceases; the patient becomes restless, and, after a few hours, talkative or even delirious. The temperature, formerly perhaps under 100°, is now found to have risen to 105° or 106°, and, unless measures be quickly taken to reduce it, it rapidly reaches 107°, 108°, even 110°, and 111°. The delirium is at first moderately active, the muscles twitching, the eyes restless, and the patient may try to get out of bed; the face is generally dusky red, and the tongue dry and tremulous. When the temperature exceeds 107° the patient becomes semi-comatose, or even quite comatose, a condition which is always present if the thermometer marks 110°.

FIG. 30.

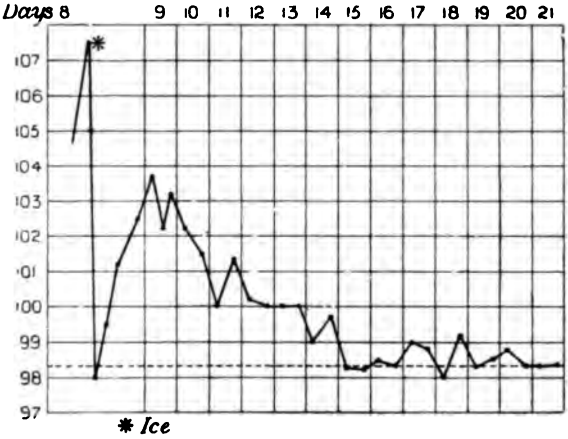


Hyperpyrexia in Acute Rheumatism.
Fatal Termination.

Under suitable treatment for reduction of the temperature, recovery often takes place; but otherwise the respiration becomes more frequent and shallow, the face more dusky or livid, the pulse rapid and feeble, râles accumulate in the chest, and death ends the scene, often within twelve or twenty-four hours of the first indication of hyperpyrexia. Even if the temperature is reduced to the normal it may rapidly rise again; and this alternation may occur five or six times, either to end in recovery or in death by exhaustion.

Course of Acute Rheumatism.—Acute rheumatism is in itself by no means a fatal disease; the majority of cases recover. If untreated, the symptoms may last from ten to fourteen days, when they will often subside; if treated by the method now usual, the pains and fever are often gone within a week. In any case, however, rheumatism shows a great tendency to *relapse*, the joints being affected in a precisely similar manner after an apyretic painless interval of from two days to a fortnight. In such a relapse the patient runs just the same risk in regard to the heart, and to the occurrence of hyperpyrexia, as he does in the first attack. Another relapse may succeed, or irregular affections of now one, now another joint, with or without marked pyrexia. Sometimes recovery is delayed by the persistence of the inflammation in one joint for weeks or months; pain, swelling, and stiffness are prominent troubles, and the joint has ulti-

Fig. 81.



Hyperpyrexia in Acute Rheumatism.

Recovery.

mately to be dealt with on surgical principles, with rests, splints, and local treatment. Another cause of delay in convalescence has already been mentioned—namely, the rapid progress of an endocarditis, so that the patient passes at once from rheumatism into pronounced heart-disease, with murmurs of aortic or mitral disease, and failing cardiac muscle.

Death takes place in rheumatism chiefly from hyperpyrexia and from the thoracic complications, especially when endocarditis, pericarditis, and pleuritic effusion on one or other side occur simultaneously. Even from these, however, the patients not infrequently recover.

Morbid Anatomy.—The condition of the joints has already been described. In the tendon-sheaths have been found opaque serum and greenish-yellow lymph. In cases dying with thoracic complications are seen the characteristic lesions described under Endocarditis, Pericarditis, and Pleurisy. Where hyperpyrexia is the cause of death, there are not necessarily any lesions, other than such slight changes as may be recognised in the joints. The lungs are mostly congested, and so are other organs, but beyond this they may be perfectly healthy; there may be no pneumonia, no pleurisy, no endocarditis or pericarditis. When lesions are present, the most common are pericarditis, endocarditis, pleurisy, pneumonia, and softening of the liver, spleen, or kidneys. Meningitis was found in two out of twenty-four cases noted in a report on the subject by the Clinical Society. In some cases of rheumatism with purpura, the intestine has presented an extremely congested and ecchymosed mucous membrane.

Pathology.—The opinion has long been held that rheumatism is due to some *acid poison* circulating in the blood. Gout, which has many similarities, is known to be associated with excess of uric acid in the blood; and the sour-smelling sweat of rheumatism seemed itself to suggest such a view. Some have held that lactic acid was the offending ingredient; apparent support is derived from the fact that Sir W. Foster caused rheumatoid symptoms by giving lactic acid to patients suffering from diabetes. Latham provided an elaborate theory of the formation of lactic acid from the decomposition of albumen, and the prevention of this process by salicylic acid, which is known to cure rheumatism. But there is no excess of either uric acid or lactic acid in the blood of rheumatic patients.

Many facts have been supposed to point to an origin in the *nervous system*, such as its association with chorea, the occasional transmission of an inherited tendency, the association of joint-disease with locomotor ataxy and some other structural changes in nerve-tissues. There is, indeed, no definite lesion of the nervous system in cases of rheumatism; but Latham has tried to connect the chemical and nervous theories by the help of vaso-motor action, and the centres which control it.

The view that rheumatism is an *infective disease*, due to a micro-organism, is compatible with much in its history and associations—*e.g.*, its close resemblance to pyæmia or gonorrhœal arthritis, the implication of the serous membranes, its relation to scarlatina and to chorea, and the facts of malignant endocarditis.

Diagnosis.—This usually presents no difficulty, the occurrence of joint-pains, with redness and swelling, fever and profuse sweating, being mostly decisive, especially if it occurs in young persons with previously good health, or, on the other hand, with a previous history of rheumatism or of heart-disease. But if multiple synovitis occurs in women after confinement, it may be of a septicæmic or pyæmic nature; and under other circumstances *pyæmia* may give rise to joint-affections deceptively like rheumatism. In the latter, the inflammation flits from joint to joint; in the former, the joints once attacked only slowly recover. *Gonorrhœal synovitis* is also more persistent than rheumatism, and it is only rarely accompanied with cardiac complications, but in early stages may be readily mistaken for it, until the presence of a discharge is ascertained. Some diseases, in which the joints are not mainly involved, may for a time be confounded with rheumatism, chiefly on account of pains in the limbs, as well as fever; for instance, *enteric fever* in its earliest stage, *relapsing fever*, and *acute infective osteo-myelitis*. A careful examination of the limbs will generally show that the joints are not the only parts of the limbs involved, as in rheumatism. The diagnosis from *gout* will be given with the description of that disease.

Prognosis.—This is, for the most part, good so far as immediate recovery is concerned. Most danger is to be apprehended from the coincidence of endocarditis, pericarditis, and pleural effusions, and from the occurrence of hyperpyrexia. In the former, recovery may take place even with extensive effusions; those into the pleura are less to be feared than those into the pericardium. Hyperpyrexia is dangerous in proportion to the temperature reached before cooling measures are adopted; but repeated rises of temperature after reduction may eventually be fatal.

Treatment.—For the efficient treatment of even mild cases of rheumatism, rest in bed is absolutely necessary; in severe cases the patient cannot do otherwise than lie still. The joints should be protected from every risk of injury. Sometimes it is desirable to raise the bedclothes from the limbs by a cradle; and some local relief to the pain may be obtained by wrapping them round with cotton wool, upon which, in severe cases, a little anodyne, as belladonna or opium liniment, may be sprinkled. The diet should consist mainly of milk; indeed, if it is well borne, it may be given alone, or diluted with barley-water, lime-water, or soda-water.

The drugs now almost universally employed are salicin, salicylic acid, and salicylate of sodium. When the patient is fully under the influence of one of these drugs, the pains disappear, the redness and swelling of the joints subside, and the temperature falls two or three degrees, it may be to the normal. If the drug is then lessened or discontinued, the pains will most likely return; if the dose is maintained, the rheumatism may be practically cured from that time; but the treatment, both by drug and diet, will have to be continued for ten days or more, at the end of which time some relaxation may be cautiously allowed. The efficient dose of either salicylic acid or its sodium salt is 20 grains, and of salicin 30 grains, every three or four hours during the first twenty-four or thirty-six hours; but in less severe cases a smaller quantity may suffice. Some give a smaller dose every hour for the first four or five hours, and then diminish the frequency to every two hours. It may happen that in the course of the second day the patient suffers from headaches, deafness, tinnitus aurium, and perhaps slight delirium, which cease when the drug is withdrawn. If it is pushed, the delirium may become active or even violent. Occasionally vomiting, a slow or irregular pulse, albuminuria, epistaxis, or hæmaturia, has occurred. As a rule, the earlier toxic symptoms coincide with the subsidence of the pains; but this having been obtained, the frequency of the dose must be reduced, so as to avoid any further development of the toxic effect, and yet keep up the influence on the rheumatic disease. Thus, for a few days it may be given every three hours, then every four or six hours, finally three times a day, until, in not less than a week from the last pain or the last abnormal temperature, the drug may be stopped altogether. About this time, also, the diet may be increased by the addition of farinaceous food, and, after a few days, meat-broth, fish, and finally meat may be given. If, however, there is any return of the rheumatic symptoms, the diet must again be reduced to milk alone for a time.

Between the three drugs under consideration there is no material difference in their effect upon rheumatism. The sodium salt is generally preferred, but the occasional occurrence of delirium and of cardiac depression makes it necessary that its use in the first days of the illness should be very carefully watched. Salicin is said to produce toxic symptoms less readily, and it is less depressant. Sometimes these drugs are not so successful; the pains continue in abated form, or relapses frequently occur. Salicylate of quinine (2—6 grains) may then be useful; or recourse may be had to the old alkaline treatment—potassium bicarbonate or acetate, 20 grains every four hours; or to potassium bicarbonate with quinine.

The treatment of the cardiac complications is described elsewhere (pp. 503, 527). Prolonged rest in convalescence is desirable after such complications to diminish any strain upon the valves or cardiac muscle. Pleural effusions, even if abundant, generally subside, and as a rule need not be tapped.

The treatment of hyperpyrexia must be prompt and energetic; it consists in the application of cold externally whenever the temperature is found to be rising above 105° F. Salicylic acid and other antipyretics cannot at present be trusted to do this with sufficient rapidity, and the best method of applying cold is by immersion of the patient in a water-bath at a temperature of 80° or 90°, which may be further cooled to 70° or 60° by masses of ice, if necessary. The patient, even though comatose and apparently moribund, must be placed in the bath and kept there from ten to twenty minutes, or until his temperature has fallen below 100° F. By this time he generally shows signs of returning consciousness. He should then be placed in bed; the extremities may be kept warm, but only light coverings should be placed on the body: a little brandy may be given, and the temperature carefully taken every half-hour. A rise to 104° should again be met by the bath. If at any time a bath is not available, the body may be cooled by packing it in sheets wrung out of ice-cold water, or by rubbing lumps of ice over the surface of the body until the required effect upon the temperature is produced. The fall of the temperature is often accompanied by an increase of the joint-pains, and the rheumatism may afterwards continue in the ordinary form.

Chronic Rheumatism.

This condition is sometimes the result of a preceding acute rheumatism, but at others it arises independently in persons of middle or advanced age. There are many who think it cannot be separated from osteo-arthritis, and in our ignorance of the nature of either disease, the question must be left open.

Symptoms.—One or more joints become painful and stiff, and the condition varies much: liable to be worse in the morning, and to improve as the day goes on and the joints are more moved; aggravated by cold and wet, therefore worse in the winter, and improved by the opposite conditions. The joints are tender, but they may show very little change otherwise, or there may be slight swelling, a little heat locally, and perhaps crackling on movement. The general health is not much affected unless the pain is severe and continuous. After a time there may be deformities from muscular action like those seen in gout or osteo-arthritis; and they may become permanent from secondary fibroid changes.

Morbid Anatomy.—As a rule, the joint changes are slight; there is very little effusion, but some thickening of the synovial membrane and cartilage, and later on, of the capsules and adjacent tissues.

Treatment.—The affected joints should be kept warm, and the pain is often relieved by local counter-irritation, as by painting with iodine, or friction with camphor and other stimulating liniments.

Internally, the salicylates may be tried, but cannot be relied on as they can in acute rheumatism. Potassium iodide, alkalies, and guaiacum may be employed, while the general health and the digestion should be cared for. Relief may be often obtained at the baths of Buxton, Bath, Harrogate, and the Continental spas mentioned under osteo-arthritis.

OSTEO-ARTHRITIS.

Under this name will be described the disease which has at different times been called *rheumatic gout*, *chronic rheumatic arthritis*, *rheumatoid arthritis*, *arthritis deformans*, and *nodose rheumatism*.

Ætiology.—Osteo-arthritis occurs at all ages. The female sex is more liable than the male. It is more common in the poor and weakly than in those of better position; but the latter are by no means exempt from it. As to antecedents, in a small number of cases there have been one or more attacks of acute rheumatism. Sir A. Garrod thinks it has association with phthisis; and Ord traced a connection with ovarian and uterine irritation in women, showing that attacks might be determined by the occurrence of the menses, or by their climacteric cessation, or by other disturbances of the function. Cold and damp often lead to an attack, as well as predispose to it. Evidence of a bacterial origin is coming forward, as organisms have been found in the synovial fluid of the diseased joints.

Morbid Anatomy.—In the early stages there may be effusion into the joint, the synovial membrane is vascular and thickened, and the articular cartilage undergoes softening. This softening begins with proliferation of the cartilage cells, and the matrix splits into fibres perpendicular to the articular surface; then the cartilage cells burst into the joint, and leave a soft ulcerated surface. By a continuation of this process the cartilage is completely destroyed, and the bony surfaces come into contact, any effusion that may be present becoming absorbed. The adjacent surfaces, from continued friction, acquire a dense, hard, white surface (*eburnation*), and may be more or less grooved or fluted on the surface; further, what was formerly a rounded or convex surface becomes flattened down, and considerable atrophy of the head and neck of a bone (*e.g.*, the femur) may in this way take place. At the same time, deposits of cartilage form along the edge of the articular surfaces growing into the capsular ligaments; and these deposits subsequently calcify. They may thus form a kind of ridge or "lip" around the joint, or in some cases (*e.g.*, in the knee) may contribute to produce large plates of calcareous matter, which surround the joint like plates of armour. The synovial membrane also forms large fringes, which may in like manner be invaded by cartilaginous deposits.

Symptoms.—In the first stage of the disease in its most pronounced forms the joints of the hands and fingers are swollen, painful, tender on manipulation, and stiff on movement. The skin is shiny and slightly reddened. These symptoms may develop in a subacute form, and subside and recur from time to time, each recurrence leaving more change behind it; or the disease may be more chronic from the commencement. The stiffness is often most marked in the morning, so that movements are at first painful; yet if they be persevered in, the pain will gradually wear off. Similar symptoms may be noted, in different cases, in other joints, such as the wrist, elbow, and shoulder, the hip, knee, ankle, and foot. The sterno-clavicular articulation, the joint of the jaw, and the vertebral joints may be also involved. Sometimes effusion can be readily recognised, and the joint is tense and elastic. In other joints, or in later stages, a peculiar creaking or grating can be elicited on passive movement. As time goes on, movement becomes more and more limited from destruction of the articular surfaces, and the development of osseous structures in the ligaments and tissues round the joints; and, finally, a fibrous ankylosis may result.

Very characteristic deformities take place in consequence of the joint-changes and the muscular atrophy which is associated with them. Thus, the fingers, instead of remaining in line with the metacarpal bones, deviate to the ulnar side, and the joint at the base of the index finger is often greatly swollen: the metacarpo-phalangeal joints are commonly flexed, the first phalangeal joints are over-extended, and the second are flexed. But other forms occur in which these joints are differently placed; and these varieties probably depend on the extent to which the interosseous and other muscles are affected. The lower ends of the radius and ulna project at the back of the wrist. The muscles commonly observed to be atrophied are the interossei in the hand, the muscles at the lower end of the femur, and the deltoid over the shoulder-joint. It has lately been stated that the nerves in connection with these joints undergo chronic neuritis.

In the hip-joint, which is often affected alone, the pain and stiffness are followed by very limited movement, apparent shortening of the limb, flattening of the buttock, and eversion of the limb. The knee and the joint of the jaw are others that may be alone involved.

When the disease arises in old people, it is liable to attack one joint, and to cause very extensive changes; whilst in the young it often attacks many joints, which are less severely diseased. But the nature of the articular lesions is essentially the same. *Heberden's nodes*, the small nodules or knobs which may form at the sides of the terminal phalanges, are regarded as an early form of the disorder.

In some cases the exacerbations of arthritis are accompanied by constitutional disturbance and pyrexia. But more often fever is entirely absent, and the illness is characterised by anæmia, weakness, and indifferent appetite. There is also a tendency to the irregular deposition of pigment in the skin.

There is considerable variability in the local symptoms—they may subside for a long period, and then burst out again; they can be relieved by treatment, but complete cure is uncommon, and an aggravation of the complaint is almost certain to occur at some time or other. In most cases there is no cardiac complication, but endocardial murmurs are occasionally heard. The disease is not in itself fatal.

Diagnosis.—Chronic *gout* is generally distinguished from osteo-arthritis by the history of the first acute attacks, and the presence of urate deposits (tophi) in the ears and in the affected joints; if necessary, the blood may be examined for uric acid. As compared with *chronic rheumatism*, we may be confident we are dealing with osteo-arthritis if the joint changes are extensive with signs of erosion of cartilage, and growth of bone, and if the disease occurs in a young person independently of preceding acute rheumatism. Arthritis affecting one joint only may be confounded with more surgical forms of lesion. The joint-changes accompanying *locomotor ataxy* generally begin with an abundant painless effusion into the joint, and there is subsequently great mobility, with destructive changes. They may be distinguished by these features, or by other evidences of locomotor ataxy—namely, Argyll-Robertson pupil and loss of knee-jerk.

Treatment.—The treatment of osteo-arthritis must be general and local. The diet should be carefully ordered, so as to avoid indigestible articles, but should not be stinted; indeed, the patients require, as a rule, good feeding, and meat may be given freely, as well as vegetables. A moderate use of alcoholic drinks may be allowed. The patient should be well clothed in flannel, a warm dry atmosphere should be looked for, and changes of temperature avoided. Various health-resorts and spas fulfilling these requirements have been found beneficial, such as Buxton, Bath, and Strathpeffer, at home, and Aix-les-Bains, Aix-la-Chapelle, Baden-Baden, and Wiesbaden, abroad. Internally, arsenic, sodium salicylate, iodide of iron, and cod-liver oil are most valuable, and they must be continued, with such intermissions as may be desirable, for weeks or months. The arsenic should be given in full doses.

Locally, much benefit may be derived by the application of tincture of iodine, small blisters frequently repeated, frictions with stimulating liniments, and passive movements, burying the joint in hot sand, dusting it with flowers of sulphur and wrapping it in flannels, or galvanising it with the continuous current at least once daily.

Any method that is adopted must be persevered with for some weeks before being given up as of no value.

GOUT.

In its typical form this is an acute arthritis, associated with the deposit of sodium biurate in the joint affected, and with the circulation of an excess of uric acid in the blood. In the majority of cases the feet, and especially the great-toe joints, are first attacked, whence the classical name *podagra;* but the joints of the hand (*cheiragra*) and other articulations are subsequently, and much less commonly even first, affected. To the excess of uric acid in the blood are attributed certain symptoms in various parts of the body, and some morbid conditions of the organs, which occur more or less alternately with the articular inflammations. For these the terms *atypical*, *irregular*, *metastatic*, *retrocedent*, and *visceral* gout have at different times been used. Of the visceral affections the most important is that form of chronic nephritis which is known as gouty or granular kidney (*see* p. 806).

Ætiology.—Gout is well known to be strongly hereditary, so that the descendants of a gouty stock are liable to outbreaks of the disorder at an early age and with comparatively little exciting cause. It is more common in men than in women, and is a disease of middle life or advanced age, though it does occasionally, in the hereditary cases just referred to, appear as early as the age of twenty; and it has been seen in boys who were only eight, nine, or twelve years old. It has been often regarded as a disease of the rich, from which the poor escape; but this is by no means entirely true, as it is not at all uncommon among hospital patients and others in poor circumstances to see the disease in its most typical form. The influence of wealth is related to diet, which is the most important ætiological factor; the ingestion of large quantities of food, especially of a highly nitrogenous kind, with abundance of alcoholic liquors, directly contributing to that condition of blood which is the essence of gout. Of alcoholic beverages, malt liquors and the stronger wines like port and sherry seem to be more prejudicial than distilled spirits. The effects of dietetic excess are aggravated by a sedentary life; and, as a rule, an occupation is prejudicial in proportion as it tempts to one or necessitates the other. But among occupations those especially must be mentioned which, like house-painting, type-founding, &c., expose the operatives to lead-intoxication, since the presence of this metal in the system, which of course may be produced in other ways (*e.g.*, impure drinking-water), certainly seems to favour the development of the disease. In those who are predisposed to it or who have already had manifestations, an attack may be brought on by an aggravation of the dietetic excesses, or by any departure from the strictest regularity hitherto found necessary; by anxiety and mental worry; and sometimes by injuries.

Symptoms.—*The Gouty Attack.*—In the majority of persons gout first shows itself by an attack of acute inflammation in the metatarso-phalangeal joint of one great toe. Various premonitory symptoms are noted in different cases; in some it may be an unusual feeling of health or exhilaration; but more often they are such as the following:—Mental depression; disturbed sleep; odd sensations, itching, or cramps in the limbs; tinnitus aurium; salivation, gastralgia, vomiting, or flatulence; alterations in the quantity and colour of the urine, which is mostly scanty and loaded with urates. These symptoms may have been troublesome for a day or two, when the patient is awakened, commonly about two o'clock in the morning, with pain in one great toe. The pain becomes worse and worse, and the patient finds it impossible to get ease. At the same time there may be a little chill, or even a rigor, and some fever. After some hours of excruciating pain, this at length abates, and the patient may fall off to sleep; when he awakes again he finds the affected joint red and swollen. It is exquisitely tender, the skin is tense and shining, and, if it can be touched, pits slightly on pressure. The veins around it are slightly distended. During the day the patient may be free from severe pain, but towards evening there is a recurrence of all the early symptoms—that is, of severe pain with some febrile reaction—which remit towards morning, to return again the following night. The joint continues swollen, and the swelling extends in the cutaneous tissues some distance up the foot; the colour is a dull, dusky red. When at length the inflammation subsides, which it does in from five to ten or fourteen days, the skin desquamates in large thick flakes, and gradually assumes its normal colour. In exceptional cases one or two toe-nails may be shed; on the other hand, in mild attacks desquamation does not occur.

The general condition of the patient is one of slight febrile reaction, with more or less gastric disturbance. The temperature is not much raised; the thermometer may reach 101°, but is rarely so high as 102°, and then only for a short time. The tongue is thickly furred, and the patient has no appetite, much thirst, nausea, and sense of distension at the epigastrium; the bowels are confined, and the motions are deficient in bile. Sometimes the sense of distension and the tenderness extend to the hepatic region.

The condition of the urine and of the blood is of much interest and importance. During the first few days the urine is scanty, high-coloured, and of great density, but the uric acid is diminished to half its usual quantity; as the attack passes off, the uric acid increases, may exceed its average amount, and then subsides to the normal, while the urine has again acquired its usual quantity, colour, and density. The blood, during the attack of gout, contains an excess of uric acid, which may be demonstrated by the thread experiment of Garrod. This consists in placing about a drachm of the serum of blood, drawn by venesection or cupping, in a shallow watch-glass, adding five drops of acetic acid, placing in the serum three or four fine threads, and setting it aside for some hours (thirty or forty) at the ordinary temperature. At the end of this time, if the threads be examined under the microscope, crystals of uric acid will be found to have formed upon them.

When an attack of gout is at an end, the patient often feels better than he has done for a long time before; and he is, as a rule, free from any reminder of his condition for a period of several months, or even two, three or more years. His second attack may be in the same joint as the first, an almost exact reproduction of it; or it may occur in the opposite foot, or in one ankle, or in the wrist or hand. A third attack often comes at a somewhat shorter interval than that between the first and second; and the periods of repose diminish in length as time goes on. Ultimately a great many joints have been at one or other time affected, and with repeated attacks they undergo changes which result in considerable deformity, so that the old gouty subject becomes crippled in somewhat the same way as the sufferer from osteo-arthritis.

When the disease has reached this stage it is in reality a *chronic gout.* If the small joints of the toes and fingers are often first affected, ultimately all the joints of the extremities may become the seat of gouty deposit, the shoulder and the hip least frequently. In the hand the joints are enlarged, more or less fixed in different positions of flexion or extension, and, it may be, with that deviation of the fingers to the ulnar side of the hand which is so common in osteo-arthritis. Similarly the foot may be fixed in a condition of talipes, or the knee or elbow in a flexed position. The swelling about the joints is often contributed to by the existence of white deposits called *tophi*, which at first lie close under a thin shining skin, with dilated venules. But subsequently the skin may yield, and the creamy or chalk-like deposit may escape in small quantities at a time; or, more rarely, suppuration takes place around the deposit, and leads to its more rapid elimination. These tophi are not confined to the affected joints; they are seen under the skin of the fingers adjacent, in the bursæ (for instance, over the olecranon), in the tendons, and with considerable frequency in the cartilage of the helix of the ear. If the creamy juice from one of these deposits be examined under the microscope, it will be found to consist of innumerable minute acicular crystals, which are composed mostly of a biurate of sodium, with a small proportion of urate or phosphate of calcium, and chloride of sodium.

Irregular Gout.—The disturbances included under this head are very various, and consist of inflammatory lesions and functional disorders in many organs in the body. The former include gastric and intestinal catarrh, bronchitis, conjunctivitis, iritis, gouty urethritis (which is, according to Ebstein, a prostatorrhœa), phlebitis, and neuritis (Buzzard). According to some authors, cirrhosis of the liver may result from gout, independent of the alcohol which is so frequently indulged in by gouty individuals. Chronic nephritis (granular kidney) has already been mentioned: it is a frequent sequel of chronic gout, but the kidneys have been found in a condition of granular nephritis with uratic deposits, while the joints were entirely free; and probably in other cases the kidneys are affected before the joints (primary renal gout, Ebstein). Some diseases of the skin, especially chronic eczema, are seen in connection with gout. On the side of the circulation, arterio-sclerosis frequently results, and fatty degeneration of the heart occasionally. Amongst the functional disorders may be mentioned migraine, vertigo, attacks of asthma and angina, muscular cramps, and lumbago. These various conditions, as a reference to other chapters will show, are not exclusively the result of gout; and their characters, when associated with gout, are not materially different from those they present when due to other antecedents.

Anatomical Changes.—If one examines after death a joint that has been the subject of gouty inflammation, the cartilage will be found covered with a bright white incrustation, either in patches or more or less completely. If perpendicular sections be made of this, it is seen to be due to a deposit of minute acicular crystals of sodium biurate in the substance of the cartilage. These form in the stratum of cartilage just beneath the surface, leaving at first a thin layer quite healthy, and as the disease progresses the deposit extends irregularly and by small more or less isolated patches into the deeper parts of the cartilage. The question has been much discussed as to whether the crystals are deposited first in the cartilage-cells or in the intermediate substance; in any case the cartilage may eventually get quite destroyed and eroded down to the bone. Collections of biurate crystals take place also in the other tissues of the joints; for instance, in the ligaments, and in the cutaneous structures, so that the movements of the joints are considerably impaired; and in some cases this may be contributed to by some of those changes—osteophytes, &c.—which occur so commonly in chronic rheumatic arthritis. In bursæ, in tendon sheaths, in the cartilage of the ear, or in the skin of parts not immediately over the joints, the essential change is also the accumulation of urate crystals. In the gouty kidney minute yellowish-white streaks, which are crystals of the same substance, may be seen, especially in the pyramids. Exceptionally true gouty deposit has been found in other situations—*e.g.*, on the spinal meninges by Ollivier, and on the meninges of the cerebellum by N. Moore.

Diagnosis.—The typical gouty attack occurring at night in the great toe can scarcely be confounded with anything else. It is distinguished from *rheumatism* by the dark-red, shiny, tense swelling of one joint, the absence of general sweating, and the slight constitutional disturbance. Later illnesses implicating many joints may show a closer resemblance. There is generally a history of many previous invasions of single joints, and less fever or sweating than in rheumatism: but the condition of the joints themselves cannot be relied upon as it can in early attacks. The swelling and redness of the back of the hand in gout may be such as closely to resemble *abscess*, but fluctuation can scarcely be obtained, and the history will mostly protect against errors. *Pyæmia* may be suggested by multiple gout, but rigors would be more severe, and the constitutional disturbance more intense. In all cases the ears should be carefully examined for tophi, and, if necessary, Garrod's thread experiment may be used as a test.

Prognosis.—Gout once declared is likely to be repeated unless the conditions, dietetic or otherwise, which have led to it are altered. Acquired late in life, and properly treated, it may not materially shorten life; but it tends to granular kidney, atheromatous arteries, and cerebral hæmorrhage. The risk of these sequelæ is more or less in proportion to the frequency of joint attacks, and hereditary gout developed early in life is very likely to be fatal by uræmia or apoplexy before old age is reached.

Nature of Gout.—One essential feature of gout is, that there is an excess of uric acid in the blood and tissues. On this point there is no difference of opinion, but very various views have been expressed as to the cause of the excess, and the way in which it is related to the several manifestations of the disease, whether articular or visceral. Roberts shows that uric acid exists in the blood as a quadrurate; and that if from any cause this is in excess, or is not rapidly eliminated, it forms sodium biurate by coming into contact with sodium carbonate. The biurate is almost insoluble in blood-serum. It is probably on this account difficult of removal by the kidneys, and at a certain point of accumulation is precipitated in the crystalline form. Its precipitation is hastened by a high proportion of uric acid in the fluid containing it. An increased quantity of uric acid in the system may be due to increased production or deficient elimination through the kidneys. Garrod adopted the latter view; but the kidneys may be structurally quite healthy in the early stages. Others have looked to excessive formation of uric acid through deficient oxidation of ingested materials, through functional inactivity of the liver (Murchison), through failure of certain tissues to change uric acid into urea (Ralfe), or through imperfect metabolism of glycocine in the liver (Latham). Luff (Goulst. Lect. 1897) believes that uric acid is normally produced only in the kidneys, that it is made by them from urea probably by conjunction with glycocine, and that its presence in the blood is determined by the failure of the kidneys to eliminate it. This failure of the action of the kidney in gout he attributes to a preceding functional or organic disease, the probable seat of which is the epithelium of the convoluted tubes. He thinks that the alkalinity of the blood has little to do with the solubility of uric acid, or of sodium biurate; but that the decomposition of quadrurates is remarkably hindered by the saline constituents of vegetables, while the saline constituents of meat diminish the solubility of the biurate. The selection of particular tissues for the deposition of sodium biurate has been attributed to their low vitality, to their deficient circulation, or to their exposure to injury; and the cartilage of the great toe-joint is regarded as illustrating all these points. Roberts attributes the selection to the richness of such tissues in sodium salts (synovia, cartilage), and partly to exposure to cold. More definite changes are believed by some to precede the gouty deposit, such as the general or local disintegration invoked by Ord; or the necrosis, which, according to Ebstein, both in the cartilage and in the kidney, precedes the deposition of the crystalline salt. The acute inflammations of gout are generally referred to irritation by the deposit, and the visceral attacks to the poisonous action of uric acid. This last is, however, denied by Roberts, who believes that even visceral symptoms may be due to temporary deposition of biurate crystals. Very strong arguments have been advanced in favour of the nervous system having a large share in the development both of the disease itself and of its local manifestations. Ord thinks that the local degenerations or inflammations may set up similar changes elsewhere through reflex nervous actions. Laycock recognised peculiarities in the innervation of the individual attacked by gout. Duckworth, also, regards the disease as essentially a disturbance of the nervous system, which is often hereditary, but may be acquired as a result of all these conditions of high living and deficient exercise, already mentioned (p. 890), combined with special circumstances depressing the nervous system. He suggests that the seat of such disturbance may be in the medulla oblongata, which is known as a centre for important nutritional and other processes, and takes as evidence of his view the many nervous manifestations seen in gouty people.

Treatment.—Whatever be the theory of gout at present adopted, the objects to be attained in those who are "gouty," or have shown a liability to attacks of acute gout, are to prevent the accumulation of urates in the system, and to favour their excretion by the natural channels. These objects can be best effected by judicious diet, by exercise, and, to a certain extent, by some drugs. The quantity of food taken should always be moderate; it should not be too highly nitrogenous, and, on the other hand, very saccharine articles of diet should not be indulged in. The latter, indeed, such as sugar, sweet fruits, fresh and preserved, can be avoided altogether, and pastry is also most undesirable; but nitrogen must, of course, be taken in meat or vegetables. Of the former, fish, chicken, game, and mutton are the best; whereas veal, pork, and salted meats are not desirable. Green vegetables are generally preferable to potatoes, on account of the large amount of starch contained in the latter. In any case, personal peculiarities may be consulted, so that indigestion is avoided, and with this proviso fat may be taken in moderation. Any one with a tendency to gout will probably do better without alcohol at all. A light claret or light dry sherry is the least harmful: or a small quantity of good brandy or whisky well diluted. The stronger sherries, port, Burgundy, and champagnes must not be taken. As in diabetes, saccharin may be usefully employed to sweeten beverages such as cocoa or coffee. The advantage of exercise in promoting chemical processes in the body is sufficiently well known. It may be supplemented by the morning bath with the use of the flesh-brush, or by the occasional use of the Turkish bath, with shampooing, or more scientific massage.

The drugs that may be of use in the prevention of gout are chiefly such as aid digestion and promote a free action of the bowels. Occasional saline aperients may be desirable, but free purgation should not be often indulged in. It is common, also, to administer the salts of potassium and lithium, since the urates of these metals are more soluble than the urate of sodium. The citrate of lithium may be given in doses of from 5 to 10 grains dissolved in water.

Acute Gout.—The affected foot should be kept raised and supported on a cradle or splint if the patient is not in bed. It should be wrapped in cotton-wool, and in severe cases some anodyne application, such as belladonna liniment, tincture of aconite, tincture of opium, or a lotion of atropine and morphia may be previously applied sprinkled on lint and some oiled silk or thin gutta percha laid over all. Leeches, hot poultices, and ice applications are to be avoided. The diet must be at once restricted in younger patients to milk and farinaceous foods, but in older persons and those broken down by previous attacks it may be more liberal, but still mostly fluid and easily digestible. Alcohol should, if possible, be entirely withheld. Medicinally, colchicum has a decided effect in most cases. It may be given as wine or tincture in doses of 15 to 25 minims every six or four hours, in combination with bicarbonate or citrate of potassium, or citrate of lithium. The bowels should be kept active. If pain is very severe, morphia may be given by subcutaneous injection or internally.

Chronic Gout. This requires the general treatment sketched for those who have shown gouty tendencies; but as the system becomes more and more broken, depletory and lowering measures must be employed with more caution. Acute outbreaks may be treated with colchicum, and even less active symptoms with the same drug, in smaller doses, more continuously. Guaiacum, potassium iodide, benzoic acid, and ammonium benzoate, are also often used, and as an adjuvant tonic especially quinine. Alkaline and saline waters are of service, and more still residence at the Spas, where the influence of the waters is combined with a regulated diet, fresh air, and pleasant surroundings.

GONORRHŒAL SYNOVITIS.

This disease, also frequently termed *gonorrhœal rheumatism*, is an arthritis, which occurs as a sequela of gonorrhœa, and to a certain extent resembles ordinary acute rheumatism. It begins at an interval of fourteen days to three or four weeks from the commencement of the urethral discharge, sometimes while the discharge is still purulent, more often during the subsequent stage of gleet.

Symptoms. In *acute* forms of gonorrhœal rheumatism several joints are at first affected with pain and swelling, but the disease soon localises itself in one only, which is most frequently, according to Davies-Colley, the elbow, but may be the knee, ankle, wrist, or foot. There is very extensive redness, with swelling, pain, and tenderness. The redness often spreads up the limb far beyond the joint, and the tissues are infiltrated to a corresponding extent. This may be such that it is actually mistaken for abscess, and it may have for the same reason a closer resemblance to gout than to acute rheumatism. The pain is very severe on the slightest movement; the fever is not high. The inflammation only slowly subsides, and leaves a good deal of stiffness behind; but it does not suppurate. Cardiac complications are occasionally observed. Davies-Colley says that this form of gonorrhœal arthritis is as common in women as in men.

In other less acute or *subacute* cases the resemblance to a mild rheumatism is in some respects closer; the joints are swollen, not so red, and with less general infiltration. All the joints in the body may be affected; but the knees, ankles, and wrists are most often involved. Not infrequently there is much pain in the fasciæ, especially in the plantar fascia; and conjunctivitis and sclerotitis occur in a certain proportion of the cases. As in the acuter forms, the inflammation tends to be persistent and does not readily subside and come again, as it does in ordinary rheumatism. It lasts two, three, or more weeks, and leaves a great deal of stiffness or even fibrous ankylosis.

Anatomical Changes.—These are serous effusion into the joint, infiltration and œdema of the tissues around it, and in severe cases perhaps suppuration, erosion of the cartilages, disorganisation of the joint, and ankylosis. Davies-Colley thinks that the synovial membrane is primarily affected in the subacute cases, and the surrounding fibrous tissues are chiefly and first involved in the acute forms.

Pathology.—Organisms closely resembling or identical with the *gonococcus* have been found sufficiently often in the fluid of the diseased joints to make it highly probable that the lesion is a specific result of gonorrhœal infection, and not a part of either rheumatism or pyæmia.

Diagnosis.—The disease is most likely to be mistaken for ordinary rheumatism, until it is accidentally discovered that the patient has a discharge; or until the persistence of the arthritis in a few joints makes one suspect the nature of the case. The resemblance may be increased by a history of previous attacks; for though gonorrhœal synovitis does not itself recur after long intervals like acute rheumatism, other attacks are often induced by fresh gonorrhœal inflammation. The acuter forms of the disease may closely resemble *erysipelas*, *abscess*, or *acute gout*. The age of the patient, and the position of the inflammation, will generally exclude the latter. *Pyæmia* may be a cause of multiple synovitis after gonorrhœa; but in this case the illness is generally more severe, with rigors, and such serious complications as pericarditis, endocarditis, pneumonia, or pleurisy.

Treatment.—It is, no doubt, desirable to cure the urethral discharge as soon as possible. For the joints alkalies and iodide of potassium have been largely employed, the latter in full doses; but it is probably better to give, as Pye-Smith recommends, plenty of good food, with cod-liver oil, and iron or cinchona. Locally, the joints should be blistered, or painted with iodine. In acute cases the limb should be kept completely at rest by means of a plaster of Paris splint; and anodyne applications, especially the compound mercury ointment, with extract of belladonna, may be used. As soon as the inflammation has subsided, the tendency to fixation must be met by friction, shampooing, and passive movements.

RICKETS.

(*Rhachitis.*)

Rickets is a disease involving the nutrition of infants and young children, of which the chief result is that the bones become enlarged at their epiphysial ends, and so soft that they are bent by the pressure to which they are put in the ordinary use of the limbs.

Ætiology.—It is essentially a disease of children, the majority of cases commencing between the ages of twelve months and two and a half years. Some cases are seen even under six months of age, the disease may even be present at birth (congenital rickets). It is not hereditary. It affects the sexes about equally. The children of the poor are much more frequently affected with it than those of the richer classes, though the latter are not exempt. This is no doubt related to what is regarded by common consent as the chief element of causation—namely, defective hygienic conditions, especially in the matter of food and air-supply. The natural food of the infant is the mother's milk, and a child should be nursed entirely until it is nine months old. The faults of diet from which infants suffer are—(1) a deficiency of quality of milk from ill-health or malnutrition of the mother, or from lactation being continued far into the second year; (2) the substitution for the mother's milk of various "infants' foods," of which the larger number contain a high percentage of starch, and the necessary limitation of the very essential fatty and proteid elements; (3) the ingestion, through carelessness or ignorance of the parents, of meat, bread, and potatoes, or other adult foods, either alone, or in addition to the mother's milk, or the artificial substitutes. Overcrowding in close unventilated rooms, and confinement to the house, may also operate deleteriously in the growth of infants. There can be very little doubt that in these several deficiences lies the cause of rickets in the great majority of cases, and though they may all have some share in it, the results of treatment are in favour of its being largely due to insufficiency of fat, and perhaps of proteids. It does not seem impossible that a defective supply from the mother to the fœtus *in utero* might start the disease even before birth. M. Parrot insisted that rickets was a manifestation of congenital syphilis, but there do not seem to be sufficient grounds for such a view.

Symptoms.—Early in the complaint some general symptoms occur, which may not attract much attention. (1) The child is restless at night, kicks off its clothes, and lies with its legs and arms exposed. (2) When it goes to sleep it perspires profusely about the head and neck, so that the pillow is saturated. (3) It is very tender about the limbs, so that it does not like to be dandled about, and even screams when it is merely touched. The first evidence of changes in the bones is seen in the *enlargement* of the *epiphysial* ends of the long bones: this is well marked at the wrists, where the end of the radius and ulna are thickened, and at the ankles and at the knees; but it is perhaps most unmistakable at the junctions of the ribs with the costal cartilages, where a series of nodules are formed, reaching on either side from the first rib near the sternum downwards and then outwards to the twelfth rib in the flank. This has been called *beading* of the ribs, or the *rickety rosary.* The defects of ossification are seen in the skull, where the fontanelles are large, and may not close until long after the usual time, which may be put at about eighteen months. Another symptom is the *delay in the eruption of the teeth*, the first of which may not appear until the eleventh or twelfth month, instead of the sixth or seventh; and the order of their appearance may present many irregularities.

Accompanying the enlargement of the ends of the bones there is an abnormal softness, in consequence of which the bones yield to the traction of the muscles or the weight of the child's body, and become bent so as to produce characteristic deformities of the limbs, the chest, and the pelvis; the head also acquires a peculiar shape, though it is less easy to see how this happens. If the disease comes on after the child has begun to stand or walk, these accomplishments are given up, and the child "is taken off its feet," as the mothers are apt to explain. If the disease begins earlier, then the art of walking may not be attained until the eighteenth or twenty-fourth month. In either case the child tries to walk before the bones are completely consolidated, and the weight of the body causes the tibiæ and femora to be bent or "bowed," generally with a convexity outwards and forwards. Sometimes there is a convexity inwards at the lower part of the tibiæ, the feet being thus widely separated, and this is to be attributed to the child getting about the floor in a sprawling position, using the feet like the hind fins of the sea-lion. If, while still unable to stand, the child crawls much about the floor, the weight of the body falls upon the arms, and the radius, ulna, and humerus get correspondingly bent. In the chest, a deformity is produced by the action of the diaphragm, which sucks in the ribs at the softest part; thus is produced a wide groove on either side of the sternum. The sternum is prominent, and the upper part of the chest has a somewhat square shape; the lower ribs, however, are often expanded over the viscera, forming the upper arch of a tumid abdomen, which contrasts strikingly with the narrow chest above. The pelvis does not usually show any deformity in infancy, but in extreme cases of rickets the pelvic aperture is considerably misshapen, being mostly of an hour-glass type, and it may afterwards, in females, offer very serious obstruction to parturition.

The head, besides presenting large fontanelles and often lines of depression corresponding to the coronal and sagittal sutures, acquires a somewhat square shape, the vertex being flattened, and the frontal and lateral regions rather prominent. In pronounced cases the cranium looks very large in proportion to the face, but contradictory statements are made as to its actual size; for while some say that hypertrophy of the brain, and distension of the ventricles (hydrocephalus) are common accompaniments of rickets, it is stated by others that the head is not really enlarged, but only seems so because the facial bones are ill-developed and stunted in growth. I believe that the circumference of the skull is often abnormally large, but it does not therefore follow that the contents are also greater; since the contents of a cube are less than those of a sphere of the same superficies.

In extreme cases there is considerable stunting of all the bones, as well as the shortening by curvature; and children of eight or ten may be no taller than those of three years old. The bones are also more fragile than normal, and *green stick fractures* are apt to occur. In some cases there is enlargement of the spleen or of the liver, or of lymphatic glands, but these changes are not constant. The appetite may be very good, and many rickety children show a perfect or even excessive development of fat; if any disturbance of the stomach or bowels is present it is to be attributed to diet, which may have caused the rickets, and not to the rickets itself. On the other hand, the nervous system is seriously involved; rhachitic children are very liable to infantile convulsions, including the special form known as *laryngismus stridulus* (p. 407).

Rickets is essentially a recoverable disease in the sense that it does not directly cause death, and that the process of bone softening ceases after a time, although it may have produced deformities that are permanent. If the disease is but slight it may leave no traces in after-life, and this is probably the case with the majority of patients: the bones become hardened, and the limbs ultimately become perfectly straight. But in other cases the effects of former rickets may be seen in the big square head with prominent forehead, the curved femora and tibiæ, and the pigeon-breast of the adult. Next to fits and laryngismus the most serious result of rickets in childhood is the aggravation of bronchitic attacks which the soft state of the ribs causes. By their want of rigidity the act of coughing especially is rendered imperfect, and the secretions accumulate, to the imminent danger of the child. Death is often brought about thus: and in other cases the frequent occurrence of bronchitis, by the collapse of lung which it produces, helps in the formation of the pigeon-breast.

Morbid Anatomy.—The changes in rickets are best seen at the ends of the long bones, or of the ribs. If the swollen portion at the junction of the rib and its cartilage be divided longitudinally, it will be seen that the line between the two structures is remarkably irregular, instead of being quite straight, as it is in healthy bones. Normally, between the already developed bone and the unossified cartilage are two narrow bands, one bluish-grey—the zone of proliferation; the other of a yellow colour—the zone of ossification. These are very narrow, quite straight, and parallel. In rickets the proliferating zone is thickened, reddened by new vessels, and has thrown out processes irregularly into the cartilage and bone on either side. Under the microscope it is seen that the proliferation of cartilage-cells, preparatory to ossification, has taken place with great freedom, but with no uniformity as it does in health; that calcification has begun unduly early in some cartilage cells, whereas it is deficient in the trabeculæ of cartilage. The processes of proliferation of cartilage-cells, of deposition of lime-salts, and of formation of medullary spaces, take place not in an uniform, regular, or progressive way, but in a most disorderly manner, and with varying degrees of rapidity at different spots. Analogous changes are seen on the surface of the bone where it is formed from periosteum; here is a soft vascular layer much thicker than is normal, showing a similar activity of the early stages of transformation, and delay in the deposition of calcareous salts. The whole bone also is unusually vascular, and the contents of the medullary cavities are redder than normal.

The muscles are flabby and wasted. The blood shows in severe cases a great deficiency of red blood corpuscles, a proportionate fall of hæmoglobin, some leucocytosis, and the presence of red nucleated cells, especially normoblasts. The changes in the spleen, liver, and glands, when they occur, appear to be due to increase of interstitial connective tissue.

Pathology.—With regard to the nature of the disease, it cannot be said at present that anything is really known of the link between the defective hygienic conditions and the bone-changes and general symptoms which result.

Diagnosis.—This rarely presents difficulties. The important early signs are the sweating of the head and the dislike to being covered at night; the tenderness of the body generally, the beading of the ribs, and the thickening of the wrists. Sometimes the inability to walk may lead to a suspicion of infantile paralysis, but the limbs can at least be moved, and the deformities of the bones should give the right clue.

Prognosis.—Recovery is the rule, the bones ultimately becoming quite firm and solid; but the deformities, if considerable, will be perpetuated. The risk of life is from the complications, especially bronchitis, with collapse of lung, convulsions, and laryngismus stridulus.

Treatment.—The first essential is the improvement of the food and general hygiene of the child. It should live in well-ventilated rooms, and should be taken out in the fresh air regularly. It should be warmly and suitably, but not too thickly, clothed. If it is being nursed, it must be understood that the supply of food is good and abundant. This is not likely to be the case if the mother is delicate, or if the nursing has been continued into the second year. In the former case additional food—*e.g.*, cow's milk, diluted with one-half, one-third, or a less proportion of water, according to the age of the child, should be given; in the latter, the nursing should entirely cease. Starchy foods, for the digestion of which the infant's secretions are by no means prepared, should be excluded, and it is best to let a good cow's milk form the chief element in the diet, to which lime-water and a little cream may be added; indeed, the directions given in a former chapter should be followed (*see* p. 628). As the infant approaches the end of the first year, beef-juice, chicken-broth, or gravy may be added; and at a later age, well boiled cauliflower, a little pounded mutton, the yolk of a boiled egg, or some custard pudding. Milk should still form a large part of the child's diet, and starchy foods should be given sparingly, if at all. The most valuable medicine is no doubt cod-liver oil, which should be given two or three times daily after a meal. The dose may be twenty to thirty drops for an infant from six months to a year old, a drachm for those beyond this age. Iron is often employed, as syrup of the phosphate; and preparations of lime, such as the lacto-phosphate, are recommended, although it will have been seen that the disease is a good deal more than a mere deficiency of lime-salts in the bones. Phosphorus has been much used abroad in doses of $\frac{1}{120}$ grain once or twice daily. While the child is under treatment, and the bones are soft, it is desirable to prevent their being bent by the weight of the child's body. Walking should be forbidden, and it may be conveniently prevented by fixing to each leg a flat splint, projecting three or four inches beyond the foot; these may be removed at night. The deformities of the limbs which remain after rickets is cured may, if extreme, be treated surgically.

Fœtal Rickets and Late Rickets.

The relations to rhachitis of cases denominated fœtal and late rickets have yet to be cleared up. It would seem that the former has some resemblances to cretinism; and cases of the latter class, in which at the age of eight or ten years, the bones become soft and deformities arise like those of true rickets, may prove to be divisible into two or more groups—one, perhaps, a true rachitis, another more like the osteo-malacia of adults.

ACROMEGALY.

In this curious disease, first fully described by Marie in 1880, there is an enlargement of the bones of the extremities, and of the face (*ἄκρον*, an extremity, *μέγας*, large).

Ætiology.—It is a disease of advanced life, and affects women somewhat more often than men; beyond this no causative conditions have as yet been traced.

Symptoms.—It is a very chronic disease, and the early stages are accompanied by pains in the head or limbs, by languid feelings and weakness, and in women by cessation of the menses. Then it is observed that the hands, fingers, nose, and face are becoming gradually larger. In an advanced condition, the nose is long and thickened, the lower jaw is enlarged transversely, and projects beyond the upper jaw, while the teeth are widely separated. Occasionally the upper jaw is also affected. The skin of the face is coarse and thickened; this is well seen in the eyebrows, and the soft parts of the nose; the lobe of the ear is hypertrophied, the tongue is large, and the voice is altered. The hands and fingers are much enlarged, the hands broad, the fingers thickened like sausages, the nails coarse, broad, flat, and ribbed. The wrist is generally small, but the feet are involved like the hands. Other bones may be also affected, and there is some bending of the back (kyphosis) and consequently diminution of stature; but the long bones are not generally bent.

Associated with these changes in the bones and integuments are some suggestive internal lesions. The thyroid gland is sometimes atrophied, at others hypertrophied; the thymus gland may be persistent, enlarged, and cause dulness at the manubrium sterni; and the pituitary body has been found hypertrophied, or the subject of tumour, *post mortem*. There may be defect in vision, and this has been found in some cases to be a double temporal hemianopia, with varying degrees of optic atrophy. Glycosuria is also sometimes present. The disease lasts several years, but no improvement takes place.

Pathology.—This is very obscure. The suggestion that it is a dystrophy dependent on disease of the pituitary body, as myxœdema is related to the thyroid, still wants confirmation.

Treatment.—The various animal extracts of thyroid, thymus, and pituitary body are being tried, but so far successful results have not been recorded.

HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY.

This is a condition of enlargement of the bones, which is most obvious in the hands and feet, and occurs in cases of chronic pulmonary disease, such as phthisis, empyema, and chronic bronchitis. The fingers are longer and thicker than normal and the nails are striated, fibrous, and curved over the much-clubbed ungual phalanges. The carpus and metacarpus are less affected. The feet and toes are similarly altered, the ends of the long bones may be swollen, and there may be effusion into the joints. Only a few cases have been examined *post mortem:* in Thorburn's case there was chronic sclerosing periostitis of many bones, with erosion of articular cartilages and fluid in the joints. The explanations offered as to its cause are: (1) that it is due to septic absorption from the pulmonary secretions; (2) that it is a tubercular lesion.

MOLLITIES OSSIUM.

(*Osteo-malacia.*)

In mollities ossium bones which have developed normally become soft and fragile.

Ætiology.—It is a disease of adults, beginning mostly between the ages of thirty and forty, and it affects women almost exclusively. Little more is known of its ætiology, except that it often begins during pregnancy, and is aggravated by it; and that it has been much more often observed in some parts of the world (along the Rhine, Westphalia, eastern Flanders, and the north of Italy) than in others.

Symptoms.—The first is pain, which begins in the lower half of the spine, the pelvis and the loins; or in the feet, knees, or other parts of the lower extremities. It varies much in severity and persistency, and may wander from part to part, or remain fixed in one spot for a long time. With this the patient becomes languid, and is disinclined to do anything. In course of time a change of figure may be noticed, the patient loses height from rounding of the spine or bending of the limbs: then, some day or other, a bone breaks, with very little apparent cause, or on slight exertion. As a rule, the fracture heals but imperfectly; and subsequently other bones break. Moreover the long bones show a remarkable degree of flexibility, so that they can be bent into very strange positions, and the more superficial bones may be indented with the finger. Though the general health may be preserved at first, exhaustion at length supervenes.

The disease runs a chronic course, and generally lasts from four to six years. Death takes place often from inability to breathe on account of the softness of the ribs; sometimes during parturition from the obstruction which the distorted (rostrate) pelvis offers to the passage of the fœtus.

Morbid Anatomy.—The bones are found to be so soft that they can be cut with a knife or indented with the finger. Nearly the whole of the bone is converted into a soft greasy mass or pulpy material, except, perhaps, a thin shell of compact tissue just under the periosteum; or even there may be nothing left but thickened periosteum itself. Microscopically, the change seems to be, first, a confusion of the natural minute structure of the bone; the Haversian systems become fused together, and then absorption of the salts takes place from the innermost rings round a canal, so that the substance is gelatinous and transparent. As the same change proceeds in the rings placed farther out, the innermost rings are entirely absorbed, the lacunæ also alter their shape and size, and finally the Haversian systems are destroyed. The bone becomes spongy and porous, and the enlarged medullary spaces are filled with fœtal marrow like the splenic pulp. In this there are recent hæmorrhages and pigment resulting from former effusions of blood. Chemical analysis of the bones shows that the inorganic constituents are reduced from 68 to 30 per cent.

The change in the bones has been attributed to the solvent action of lactic acid, which is said to have been found in the bone-marrow as well as in the urine. Rindfleisch thinks carbonic acid is the solvent. The intimate connection with the ovaries suggested by its occurrence and aggravation during pregnancy, is confirmed by the fact that the disease has been arrested by the removal of these organs.

The **Diagnosis** is not difficult when the bone-symptoms have begun. Earlier symptoms may be mistaken for *rheumatism*.

Spontaneous fracture also may take place in *sarcoma* or *carcinoma* of the bones.

Treatment.—Porro's operation and the removal of the ovaries have been advocated, and several times performed. The existing deformities remain, but further progress is checked. Otherwise, recovery is exceedingly rare, and the treatment can only be tonic, as by quinine, iron, and cod-liver oil.

DISEASES OF THE SKIN.

ELEMENTARY LESIONS.

BEFORE describing the diseases of the skin it is necessary to mention the various changes taking place in its structure, which constitute the *elementary lesions* common to several different diseases. The older classification of Willan and Bateman was based upon those lesions: there was a group of vesicular diseases, one of scaly diseases, one of papular diseases, and so on. But it is now seen that such a classification brings together diseases that have no special connection; and, on the other hand, there are many diseases which produce at the same time, or successively, two or three different elementary lesions, and would be, therefore, put into as many different classes.

Hyperæmia.—This consists of redness, of varying extent, due to the blood-vessels being distended with blood. It may be—(1) an *active hyperæmia* from vasomotor paralysis; (2) an *early inflammatory hyperæmia*, with slight swelling, tenderness, and some indications of pyrexia; or (3) a *venous hyperæmia*, or passive congestion, with a more blue or livid colour than in the other forms. In all cases the redness disappears on pressure, to return when the pressure is removed—quickly in the first two cases, slowly in the last.

Hæmorrhages.—Cutaneous hæmorrhages form larger or smaller spots of bright red, dark red, or purple colour: they do not disappear on pressure; the blood is gradually absorbed, and the colour fades into brown or brownish-yellow, or goes through several changes, becoming successively brown, green, and yellow, in the large subcutaneous hæmorrhages. A yellowish-brown stain may be left for a long time. The smaller spots are called *petechiæ*, the larger *ecchymoses:* if they form streaks they are called *vibices*. In *capillary ecchymosis* a very fine mottling is produced, which looks like a hyperæmia, until it is found to persist under pressure.

Papules or **Pimples.**—Small red or pink elevations of the skin, solid, or at least not visibly containing fluid. They arise mostly in the cutis, but may be imitated by accumulations of epidermic scales.

Vesicles or **Vesiculæ.**—Small blisters (from 1 to 5 mm. in diameter), due to the accumulation of clear fluid under the upper layer of the epidermis. They are frequently inflammatory, seated upon an inflamed base, and contain a yellow albuminous serum.

Blebs or **Bullæ.**—Larger vesicles, from 5 mm. to an inch or more in diameter. The fluid contents are clear, or slightly turbid, or blood-stained. They are often situate on an inflamed base: they heal by discharge of the contents, and the drying and shedding of the epidermic scale.

Pustules or **Pustulæ.**—Vesicles or bullæ containing pus.

Scabs or **Crusts.**—Irregular flat masses of dried serum, pus, or blood, or a mixture of these materials, forming upon and adherent to the raw surface which has secreted them, and frequently the result of a vesicle, pustule, or bulla.

Wheals or **Pomphi.**—A circumscribed œdema of the corium, producing a pale pink or white elevation of the skin.

Scales or **Squamæ.**—Collections of epidermic cells in the form of flakes. Sometimes, as in seborrhœa, there is a large admixture of the fatty matter of sebum. Scales vary from the small branny particles of measles (furfuraceous) to the large exfoliations of pityriasis rubra and some cases of scarlatina, or the thick adherent masses of psoriasis.

Scratch Marks.—Linear lesions of the skin, from a third to two inches in length, produced by the nails, and bearing small crusts of blood. Ultimately, if deep enough, they become linear or fusiform cicatrices. Their direction generally bears a definite relation to the position of one or both hands, and parts of the body which the hands cannot reach are exempt from them.

Raw or **Excoriation.**—A patch of skin deprived of the upper layer of the epidermis, and exposing the rete Malpighi. It is of a vivid red colour, and tender to the touch; and it secretes a small amount of serum, which may dry into a crust.

Chaps, Rhagades, or **Rimæ.**—Cracks or fissures through the epidermis, reaching the rete Malpighi or corium beneath, very sore, and apt to bleed.

Sore or **Ulcer.**—A loss of substance involving the epidermis, and extending to the papillary layer. The base is covered with granulations, and secretes pus.

Scar or **Cicatrix.**—The new growth of connective tissue, which results from the healing of sores, involving the papillary layer and deeper corium. When recent, they are pink or bluish in colour. Finally they become dead white, and contract in size.

Nodules.—Solid elevations larger than papules. They have been called tubercles—a name which is now best limited to the specific lesion which is the first change in phthisis.

Stains or **Maculæ.**—Small areas of pigmentation deeper than that which is normal to the part. They may arise from a preceding hyperæmia, and then disappear shortly. They are generally more permanent if arising independently.

Various disorders of the skin have been already described, as, for instance, the eruptions characteristic of the exanthems, the cutaneous lesions of syphilis, lepra, glanders, actinomycosis, and elephantiasis, and the forms of hæmorrhage known as purpura.

INFLAMMATORY CONDITIONS, OR FORMS OF DERMATITIS.

ERYTHEMA.

Erythema (from ἐρύθημα, a blush) has a rather wide signification. A redness of the skin may be set up by external irritants, or form a part of the infectious diseases, called exanthems, and to these the name has been applied. In both these cases there is an early phase of inflammation. Another class has been called by Hebra *Erythema exudativum*, and is characterised by much more definite and pronounced inflammatory lesions. The skin is red, swollen, and tender. In some cases bullæ or vesicles are formed; and blood may be effused into them or into the corium. Other cases may be considered as intermediate, in which there is certainly more than mere hyperæmia; but the inflammatory exudation is not considerable. It will be best to notice, first, the more marked conditions known as Erythema exudativum, which includes E. multiforme, E. iris, and E. nodosum.

ERYTHEMA MULTIFORME.

As the name suggests, there is very great variety in the lesions produced. In all cases they are bright or dark-red elevations of the skin, which may be in the form of papules (*E. papulatum*), or in larger patches or nodules, the size of a sixpence or a shilling piece. Such a patch may clear at the centre, and leave a ring (*E. annulatum*); if this enlarges it may coalesce with neighbouring rings and produce sinuous or scalloped patches (*E. gyratum*). *Erythema marginatum* has a similar outline: the peripheral margin of the red band forming the ring is raised abruptly, and the central margin gradually slopes towards the skin. Sometimes a ring of erythema is surrounded by another ring outside it, and this by another farther out, while the first ring is beginning to fade. I have seen four such rings at the same time.

Occasionally bullæ or vesicles appear on these patches, and sometimes petechiæ or ecchymoses may occupy the centre of a broad papule. The raised patches last a few days and then gradually subside, often leaving a brown or brownish-yellow stain, even if there has been no obvious hæmorrhage into the structure of the skin. The whole duration is from two to four or six weeks. It may begin with some malaise; it is occasionally accompanied by not very definite joint-pains; and it is a not infrequent occurrence to have an eruption of erythema, especially E. marginatum and hæmorrhagic varieties, in the course of ordinary acute rheumatism. The hæmorrhagic forms are called *Peliosis rheumatica*, but hæmorrhages (purpura) certainly occur also in rheumatism without any preceding true erythema (*see* p. 760).

Erythema multiforme occurs more often on the back of the hand and forearm, the front of the leg, and the dorsum of the foot; and on the face, neck, front of the chest, and abdomen.

Erythema Iris—Erythema Bullosum—Herpes Iris.

This appears to be an erythema with the formation of vesicle or bulla as a result. It is comparatively uncommon. Two varieties are described. In the first, after some preliminary tingling, a small papule forms, which soon shows a minute vesicle upon it. The vesicle enlarges, becomes flat, and is surrounded with a pink areola. After a time the fluid is absorbed from the centre, leaving a purplish depression surrounded by the still vesicular periphery. Or the centre remains fluid, then comes a zone of purplish depression from absorption, then a peripheral zone still fluid, then the areola outside all. These zones of different colours suggest the name of iris. Sometimes complications arise from coalescence of extending patches. The recovery of any patch takes place in about a fortnight by fading of the areola, absorption of the fluid, and subsidence of the papule; but from repeated crops the whole disease may last from four to six weeks. The backs of the hands and fingers, especially the radial half, and the insteps and knees, are the parts most affected; and the disease is generally symmetrical.

In the second variety there is a central bulla, and round this a ring of vesicles of smaller size. A second ring may form round the first, and a third round that. In some of these and allied cases of bullous erythema, the contents of the vesicles may be purulent, or sanguineous, and the process may extend deeply enough into the corium to produce ulcers which are followed by scars.

ERYTHEMA NODOSUM.

This consists of oval or circular solid flat elevations of the skin, from half an inch to one and a quarter inch in diameter, bright or dusky-red in colour, gradually shading off into the surrounding skin, tender to the touch, and perhaps pitting slightly on pressure. They occur most often over the whole length of both tibiæ, and not infrequently over both ulnæ. Though rare in other parts, they may be seen on the calf, on the thighs, over the scapulæ, and over the condyles of the humerus. They come out more or less in crops, last seven to ten days, and gradually subside with bruise-like staining. They may become soft and fluctuate, but never suppurate. They are most common in children, and people under twenty years of age; and more frequent in girls than in boys. Their onset is preceded by some malaise, pains in the joints, and slight pyrexia; and there are some grounds for regarding it as an infectious disease. On the other hand it is often associated with acute rheumatism.

Pathology of Erythema.—The essential change in erythema is an inflammation of the corium, with hyperæmia and effusion of lymph and corpuscles. In different cases the process seems to be more intense at different levels in the skin structures: sometimes the skin alone is swollen, at others the epidermis is raised into blisters. The vesicles may contain either serum, pus, or blood; and the blood extravasated may be a visible hæmorrhage, or only enough to produce staining as recovery takes place.

Treatment.—There is no specific treatment for the erythemata as such. Their cause, if it can be recognised, should be dealt with, and relief to discomfort about the lesions can be given by local means. In cases having a rheumatic origin, salicylate of sodium should be given in doses of 15 or 20 grains three or more times in the day. If gastric troubles have caused the erythema, unsuitable articles of food should be withdrawn, such as shell-fish, salted fish, pork, and sweets. Bicarbonate of sodium, bismuth, and gentian, should be given; or, in other cases, acids and nux vomica, according to the character of the gastric disorder. Locally, astringent and sedative lotions are of value, especially lead lotion (liq. plumbi subacet. dil.), combined with opium if there is much irritation, or calamine lotion or evaporating lotion of spirit and water, eau de Cologne, or liq. ammon. acetatis; in the drying or scabbing stages of the vesicular forms, zinc or zinc and lead ointment. In erythema nodosum, iron in combination with saline purgatives is generally the best remedy—*e.g.*, the sulphates of iron and magnesia, with peppermint water.

ERYTHEMA PERNIO.

Pernio, or chilblain, is a superficial dermatitis, affecting the toes, sides of the feet, and the fingers, as a result of cold in people of defective circulation, and especially in children. There are patches of dusky redness, with itching, smarting, and pain, which come on frequently with the cold winter weather, and may only completely subside with the return of spring. In severe cases, or if irritated by friction or injury, they may vesicate or form indolent ulcers. They should be prevented, if possible, by warm clothing, sufficiently loose boots, and active exercise, such as running, skipping, dancing, and skating.

Treatment.—When they occur they should be rubbed with lin. camph. co., with or without lin. belladonnæ; or tr. iodi may be painted on the feet, or unguentum iodi rubbed in. Tr. iodi, decolorised with half its quantity of liq. ammoniæ, may be used for the hands (Crocker).

ERYTHEMA INTERTRIGO.

(*Eczema Intertrigo.*)

This is the inflammatory redness which occurs in the folds of the skin in fat people, especially under the mammæ, between the buttocks, and between the thighs and the scrotum or labia in children. The redness corresponds closely to the parts of skin that are in contact; the surface is raw, denuded of the upper layers of the epidermis, and it secretes a whitish turbid fluid, different from the yellow serum or sero-pus of eczema, and not drying into crusts unless mixed with the medicinal substances applied to it. In children, it is no doubt aggravated by contact with napkins wetted by the urinary and fæcal discharges; and, as it not infrequently coexists with parasitic stomatitis or thrush, the mother generally regards it as thrush, which has passed through the child. It may coexist with ordinary eczema. It must be distinguished from syphilitic eruptions, which usually spread beyond the limits of the contact of skin with skin, or skin with napkin, and may be quite dry.

Treatment.—The parts should be separated, and contact with secretions and wet napkins should be prevented. This is best done in children by speading zinc ointment on narrow strips of lint, and carefully laying them over the thighs and scrotum or labia, so that they are protected from one another and from the napkin. The lint should, of course, be changed directly it is wetted or soiled by the motions. Slight cases may be dusted with fuller's earth or zinc oxide. Any defect of health should be treated, especially diarrhœa, the acrid discharges of which may intensify the trouble. In adults, powder of zinc oxide, mixed with two or three parts of starch, or of boric acid and kaolin may be dusted over the part and a piece of lint placed in the fold; or ointment of zinc or boric acid may be used.

Other Forms of Erythema.

Erythema læve is the hyperæmia which occurs in the tense skin of anasarcous limbs. It may go on to deeper dermatitis and sloughing.

Erythematous eruptions also form part of the epidermic diseases known as *pellagra* and *acrodynia*. The former occurs in Lombardy as a result of eating decomposed or fermented maize; the latter was epidemic in Paris in 1828–29, and has since been seen but rarely.

URTICARIA.

Urticaria (*urtica*, a nettle).—This has a close alliance with erythema. The eruption often comes out suddenly, and consists of firm, round, convex, or lenticular elevations of the skin from a quarter of an inch to an inch in diameter, at first pink, and soon becoming white in the centre. These are called pomphi or wheals. They are scattered or closely crowded over the part affected, and are not symmetrical. They may arise very rapidly, and subside in a few hours or a day (*U. acuta*), or they last longer, or recur frequently (*U. chronica*). Sometimes the elevations are quite small (*U. papulata*). Rarely a small vesicle may form on the surface of the wheal (*U. bullosa*). Urticaria is accompanied with intense itching, so that the patient cannot forbear from scratching himself, and thus, no doubt, the lesion is considerably aggravated.

Causes.—Urticaria is due in a large number of cases to the action of poisonous substances, either applied locally, or circulating in the blood after absorption from the alimentary canal. Among the former we have the poison of the stinging nettle, the sting of bees and wasps, and contact with jelly-fish and certain caterpillars. U. papulata, which occurs especially in children, is perhaps often due to the irritation of fleas and bugs.

Among the latter are (α) certain foods, especially shell-fish, the less digestible meats, pork and sausages, mushrooms, and some fruits; (β) certain drugs, copaiba, cubebs, quinine, and others. In (γ) some general disorders, such as gout, indigestion not specially related to the above ingesta, menstruation, lactation, pregnancy in women, and asthma there is also the possibility of toxic substances being in circulation. But there are chronic cases in which no such cause can be traced.

In some individuals direct mechanical irritation will cause a local infiltration to take place almost at once, so that the scratch of a pen or the nail upon the skin is sufficient to raise a linear ridge; and thus figures or letters can be traced upon the skin, lasting for some minutes (*factitious urticaria*).

Treatment.—The cause must be looked for and removed. In acute cases clearly due to ingesta an emetic is indicated. In other more chronic cases, the diet should be carefully considered, the bowels should be regulated, and any defect in digestion met by suitable treatment. In gouty people, colchicum and salines should be given; and in others, general tonics may be desirable. Chronic urticaria is sometimes very intractable; quinine, with saline laxatives, or arsenic, may then be of value. The severe itching requires local treatment. Scratching must be prevented, and one of the following applications should be used:—Alkaline baths (sod. bicarb. 2 to 6 oz. in the bath), or alkaline lotions (sod. bicarb. ʒj. or ʒij. to ℥vj.), hydrocyanic acid lotion (ʒiij. of dilute acid in aq. ℥xx.), and lead lotion. Crocker especially recommends the antiseptic lotions:—Liq. carb. detergens (ʒij. or ʒiij. to aq. ℥viij.); terebene (℥j. to ℥viij.); sanitas and water, equal parts; a saturated solution of benzoic acid, carbolic acid (ʒj. or ʒij. to ℥viij.), and others.

URTICARIA PIGMENTOSA.

This is a curious eruption of which only a small number of cases is on record. It begins in early infancy, and consists of raised patches, at first of a brownish-red, later of yellowish or fawn colour. The patches may remain unchanged for years, but some after a time disappear, leaving only pigmentation. The majority of cases have been accompanied by severe itching, with the result of producing secondary wheals, factitious urticaria, and erythema.

Treatment has hitherto had very little effect, beyond the relief of itching; but the patches tend to disappear towards the age of puberty.

PEMPHIGUS.

This is an eruption of bullæ or blebs occurring as an idiopathic affection. Bullæ have already been described as occurring in some forms of erythema, and they may be caused by burns, irritants such as blisters, and by the itch-acarus; but in pemphigus, which is a rare disease, the bulla is the primary and chief lesion.

P. vulgaris may be acute or chronic; the latter is more common. The eruption may be preceded in some people by chilliness, nausea, or pyrexia; then the bullæ appear at one or other part of the body, small at first, gradually increasing in size, tense, hemispherical, with clear yellow or slightly turbid contents. The fluid is an albuminous serum, and the turbidity is due to increasing amounts of leucocytes. Around the bleb the skin is at first quite normal, but a narrow pink areola is acquired later, and increases in proportion to the opacity of the fluid. After a few days the fluid is absorbed, or the bleb ruptures, and shrinks down on to its base. From this it is subsequently shed; it leaves a mark which is injected and afterwards slightly stained but rarely or never scarred. Sometimes the bleb contains pus or blood, and after its rupture, the base may be covered with yellow lymph, or may slough.

The number of bullæ in any case is very variable. There may be but few in one part, or isolated bullæ in different parts of the body; or the whole surface may be thickly covered by blebs, which come out in successive crops, lasting only a few days each, but keeping up the disease for weeks and months. Nearly every part of the body may be affected, but the hairy scalp least of all. Rarely the conjunctiva is attacked with pemphigus, which is followed by shrinking or contraction, producing deformity and ultimately blindness.

The amount of general disturbance is greatest in children and old people, and is proportionate to the extent of surface involved. Young adults with few bullæ are not materially affected. Sometimes there is severe itching, with all the secondary results which follow scratching, such as wheals, eczema, and pustules, while the contents of the bullæ are likely to become more purulent (*P. pruriginosus*).

Acute pemphigus is much more rare than the chronic form; the course is rapid, and pyrexia is present. Most cases of bullous formation in infants are due to syphilis, but Crocker considers that there is an independent *P. neonatorum*.

P. foliaceus is a very rare and fatal form of pemphigus, in which the whole surface of the body is gradually involved. The blebs which form are flaccid and flat, never tense and hemispherical. Their contents are turbid, and when these escape an inflamed excoriated surface is left; to this the remains of the bullæ adhere, forming thin flakes, the under surface of which is moist with an offensive secretion. If the flakes are removed there remains a red, raw, secreting surface, not unlike eczema rubrum. When the whole surface is affected it is mostly covered with the adherent epidermis, and with raw patches at intervals; then also the occurrence of blebs is not easy to observe, as they form under existing epidermis and soon rupture. The course is slow, with remissions and relapses, it may be with healing of the skin in parts; but eventually the disease is fatal by exhaustion or intercurrent disease.

P. vegetans is another rare variety, in which the mouth is first affected, then bullæ of ordinary type form on the skin, ulcerate, and remain unhealed for a long time. The characteristic feature is that in moist situations, like the axillæ, groins, and gluteal folds, fungating papillary growths form on the site of the ruptured blebs, project a quarter to half an inch above the surface, and secrete an offensive muco-purulent fluid. Severe prostration ensues, and the cases end fatally.

Ætiology.—Pemphigus vulgaris occurs at all ages, but is more common in children than adults, and in females than males. Heredity has been noticed a few times, but beyond this very little is known of its causation. P. foliaceus has been referred to chills; it sometimes supervenes on P. vulgaris.

Pathology.—There is inflammation of the papillary layer of the skin, with fluid effusion lifting the epidermis, but whether the bleb forms in the layers of the rete or below seems yet uncertain. Diplococci and staphylococci have been found in the lesions of pemphigus, but their relation is not yet clear. Crocker points to the occurrence of bullæ in diseases of the central nervous system as suggesting a possible pathology for pemphigus.

Treatment.—Arsenic has long been advocated by Hutchinson as a specific in pemphigus, and its very great value has been allowed by most observers. It must be given perseveringly in full doses. Its value in acute cases is not so certain. Locally, some relief from discomfort may be obtained by the use of zinc ointment, zinc oxide dusted on, or similar soothing applications. Tonics, quinine, iron, &c., and sufficient food are desirable also. P. foliaceus yields to no drug, and P. vegetans seems equally intractable, but opium is recommended for trial.

HERPES.

This name has been given to certain vesicular diseases, but it is not easy to give a definition that will cover all. One may say that the vesicles of herpes are generally smaller than those of pemphigus, are seated upon an inflamed base, and terminate by scabbing. The diseases for which the name is still retained are herpes zoster or zona, herpes labialis, and herpes preputialis.

ZONA.

(*Shingles.*)

This is an eruption of vesicles, arranged in groups, which always correspond in position to the distribution of a cutaneous nerve. The name zona, or girdle, is taken from the most common or intercostal variety, in which the groups of vesicles extend from the spine round one half of the body to the middle line in front. The eruption is preceded sometimes by pain, tingling, or smarting, and it may be a little malaise; then appear groups of closely set papules, forming red patches, one or two inches in diameter; and upon these the vesicles quickly arise, with thin walls, clear contents, not very tense, and, when numerous, acquiring a polygonal form from mutual compression. The patches do not all appear simultaneously—for instance, one may form first near the spine, then later one in the axilla, and later again one near the sternum; some patches also—that is, the later ones—may fail to produce any vesicles, the process, as it were, subsiding early or aborting. After a time the contents of the vesicles become opaque or milky, and the vesicle dries into a scab, which drops off, leaving a red stain. The milkiness may amount to the formation of pus, and the superficial layer of the skin may be destroyed, so that scars result. Scars may form in each group, but not in every vesicle of a group. Quite rarely extensive sloughing of the skin takes place, leaving deep ulcers, which heal slowly. The patches are distributed in the course of a nerve: on the trunk they form a band two to four inches broad, generally somewhat more horizontal than the true course of the ribs, and the vesicles may transgress the middle line both in front and behind.

H. frontalis occupies the area of the supraorbital nerve on the forehead and scalp, and there is often conjunctivitis. *H. cervicalis* lies over the neck, clavicle, and deltoid; *H. brachialis* follows the course of the nerves of the arm; and other similar groupings on the abdomen, thigh, and leg, are occasionally seen. An intercostal zona may be accompanied by herpes of the inner side of the arm (intercosto-humeral nerve), or a gluteal by an anterior crural, representing posterior and anterior branches of the lumbar nerves. The eruption is nearly always unilateral, and its bilateral occurrence has very rarely been recorded.

The duration of the eruption is from four to ten days, but the disease does not always end here. Especially in old people, neuralgic pain in the course of the affected nerve may continue for months or years, and be a source of serious trouble; and in a few cases paralysis of associated motor nerves or nerve-fibres has been seen, most often of the seventh or third nerve in facial zoster, but also of the nerves to the deltoid and abdominal muscles.

Pathology.—The eruption itself is an inflammation of the papillæ and corium, followed by effusion into the layers of the rete; but the curious relation to cutaneous nerves has naturally led to investigations as to their condition, and in a great number of instances lesions have been found. The most frequent is inflammation of the ganglion on the posterior root of the spinal nerve, and of the nerve below it; but other changes are recorded, namely, inflammation of the sensory root above the ganglion, peripheral neuritis, neuromata, and hæmorrhage into the Gasserian ganglion.

Diagnosis.—This depends on the unilateral groups of vesicles, corresponding to the distribution of a nerve.

The **Prognosis** is favourable, but the probability of scarring, and the tendency to troublesome neuralgia in elderly patients, must be remembered.

Treatment.—Little is required except to protect the vesicles from injury, and rubbing by the clothes, and to allay any irritation, tingling, &c. This may be done by the application of zinc ointment, lead lotion, or powdered zinc oxide, with the addition of a little morphia, if the pain is severe. For the severe pain afterwards arsenic should be tried. Fagge thought he saw benefit from vinum colchici. Locally, menthol may be rubbed in, or morphia injected. Blisters over the origin of the nerve, and the continuous current, also give good results (Crocker).

HERPES FACIALIS, OR LABIALIS.

This occurs as groups of vesicles forming rapidly upon an inflamed base. The vesicular contents are clear at first, then turbid, and afterwards dry into a scab, and fall off, leaving scarcely a mark. The process lasts from five to ten days. It affects the lips, the alæ of the nose, and the adjacent cheeks, and is usually bilateral. It mostly occurs in association with some acute febrile disease, especially with croupous pneumonia, of which it is sometimes considered diagnostic; but this is not so, as it happens in ordinary catarrh and in bronchitis. I have seen it in relapsing fever, and one sometimes gets it without any other recognisable disease, other than a sharp pyrexial attack, with high temperature and rigor. It not infrequently recurs in the same person. Local sedative applications, such as zinc lotion or calamine lotion, are all that are required.

HERPES PREPUTIALIS.

This closely resembles the preceding. It consists of a vesicle or a group of vesicles on an erythematous base, occurring on the inner side of the prepuce, less often on the outside, on the glans, in the meatus, or even in the urethra. But it is seen sometimes on the labia, nymphæ, and mons Veneris in women. It is often preceded by some local disease, such as gonorrhœa, soft chancre, or stricture of the urethra, and it is of importance, because the vesicles rupture early, and form small ulcers, which may be mistaken for chancres. Like herpes facialis it is apt to recur. The treatment is to keep the parts thoroughly clean, and to apply lead lotions on strips of lint, or dust starch and zinc oxide over the vesicles, and separate the parts with lint. Iodoform, or lotio nigra on lint may be used where a sore has formed.

DERMATITIS HERPETIFORMIS.

Dühring, who introduced this term, gives the following definition of the disorder to which he applies it:—"An inflammatory, superficially seated, multiform herpetiform eruption, characterised mainly by erythematous, vesicular, pustular, and bullous lesions, occurring usually in varied combinations accompanied by burning and itching, pursuing usually a chronic course with a tendency to relapse and recur." This includes cases hitherto called *hydroa*, probably some of the cases recorded as pemphigus, erythema iris, and pemphigus iris. It also covers *herpes gestationis* or *pemphigus gestationis*, a bullous eruption which occurs during pregnancy, affecting more or less the whole of the body, disappearing with delivery, and recurring generally in future pregnancies. D. herpetiformis thus presents the most varied lesions over the body at the same time, in one place patches of erythema, in another urticarious wheals, and in a third bullæ like those of pemphigus; and there may be pyrexia with it. The disease is very little amenable to treatment; the lesions may spontaneously heal at one part, and break out in another.

The lesion is an acute inflammation of the papillary layer of the corium with the formation of vesicles directly beneath the epidermis; and the extravasation of enormous numbers of multinuclear and eosinophilous leucocytes. The proportion of eosinophile corpuscles in the blood is also largely increased.

Treatment.—Arsenic is of value internally, and locally ichthyol preparations (*see* p. 926) may be employed.

CHEIROPOMPHOLYX.

This name, meaning bullæ on the hand (from χείρ, a hand, and πομφόλυξ, a bubble) was given by Hutchinson to an affection which is not very common, at least in its most marked stages. The subjects are young people, more often women who are slightly out of health, and who are inclined to perspire about the hands; it occurs also mostly in the heat of summer. After a little tingling and itching there appear small translucent spots on the skin of the sides or tips or bases of the fingers, and in the palm of the hand. These spots look like, and indeed are, vesicles beneath the horny layer of the skin, which, however, is not raised by them until they reach a very great size. They may be isolated, but when abundant form groups of half an inch to an inch or two in diameter of closely packed vesicles, so that the affected skin, still flat, looks as if it were formed of boiled sago grains. If one of these is punctured a clear, or very slightly turbid, perhaps ropy, fluid exudes, which is neutral or alkaline, and soon, if not at first, contains albumen. There is no reddening of the skin unless just at the margin of a large patch. In later stages the septa between the vesicles may disappear. Several vesicles run into one another, and the horny epidermis is raised into large blebs or bullæ projecting beyond the skin. After a week or more the contents are absorbed, and the skin forms a dead flake, which is shed; it leaves a pink new skin, which gradually assumes the normal appearance. The only other parts of the body subject to it besides the hands are the feet, and they are not always, and never so badly, affected.

Anatomically it is seen that the vesicles form in the rete often in connection with the sweat-ducts. Tilbury Fox regarded them as retention cysts, and called the complaint *dysidrosis;* but the serous nature of the fluid, and the presence of leucocytes in the subjacent papillæ show that the lesion is an inflammation of the papillary layer of the epidermis.

The patient, as a rule, readily recovers, but the disease is liable to recurrence. Its recognition is not difficult; the curiously aggregated vesicles within the skin are characteristic.

Treatment.—A tonic treatment should be adopted with the local applications of sedative lotions, such as those of lead and zinc.

ERUPTIONS PRODUCED BY DRUGS.

A number of very different drugs produce eruptions as a result of their internal administration. Such an eruption is generally some kind of dermatitis; it may be erythematous, urticarial, vesicular, bullous, purpuric, or in some other form, but the first four varieties are more common, and especially the first—namely erythema. The most important of these eruptions will be shortly noticed.

Antipyrin.—A red, papular or morbilliform eruption over the greater part of the body, sometimes with itching and subsequent desquamation. Purpura has been also seen.

Arsenic. — Urticaria, erysipelatoid rash, or small papules. Zoster has occurred during the use of arsenic, but the relation of the two has not always been obvious. The long-continued use of arsenic has caused a general pigmentation of the skin, and in psoriasis the healed patches sometimes become very deeply stained.

Bromides. — An acneiform eruption is common as the result of the use of bromides in epilepsy—the pustules are commonly discrete, and occur on the face, chest, back, or scalp, and around the hair follicles on the thigh. More extensive lesions occur in exceptional cases in children on the face and limbs; these are large, oval or circular, much raised patches of deep red colour, covered with a number of pustular points, or the thick scab which follows their rupture. The substance of the patch is soft; it mostly subsides, and the scab is detached, without leaving any scar, but only a rather persistent stain. The lesions often begin some days after the bromide has been stopped, and their appearance is favoured by disease of the kidneys hindering elimination of the drug. Arsenic internally promotes their cure, and if given with the bromide tends to prevent their occurrence. Erythematous, papular, and bullous eruptions also occur.

Iodides.—The eruptions are erythematous, pustular, vesicular, bullous, or purpuric (*see* p. 759). The erythema is papular, and occurs over the trunk, face, and limbs. Pustules are seen like those of the bromide rash, but smaller in size when discrete; and the confluent forms are less common, and tend to be more bullous. Sometimes large bullæ occur, with a very narrow areola around them, and clear serous contents. Like the bromide eruption it may be delayed for some days after the drug has been stopped, and is more likely to appear if the kidneys are diseased. The addition of arsenic, or aromatic spirits of ammonia to an iodide mixture, or taking the dose in half a tumblerful of water, may be tried to prevent its occurrence.

Chloral. — Erythematous eruptions, diffuse redness or red papules, and occasionally purpura. They occur mostly after long-continued use of the drug.

Chrysarobin.—Goa powder and chrysophanic acid, when applied to the skin for tinea or psoriasis, produce a dusky copper-coloured redness, with œdema or tenderness, far beyond the limits of the application.

Copaiba. — Erythema, consisting of bright-red, roundish or irregular patches, slightly raised above the surface, here and there confluent, somewhat like measles, covering the arms, legs, trunk, and face. Purpura, vesicles, and urticaria are occasionally present. Desquamation may occur after a persistent eruption.

Cubebs seem occasionally to produce a similar rash.

Quinine. — Erythematous rashes are most common, either diffuse or papular; an urticarial form is next most frequent; both of these produce severe itching, and erythema may be followed by extensive desquamation. Purpuric, vesicular, and bullous rashes are less often seen.

Other drugs that have more or less frequently caused rashes, mostly of an erythematous or urticarial type, are belladonna, boric acid, cannabis indica, potassium chlorate, chloroform (inhalation), cod-liver oil, digitalis, iodoform, mercury, morphia, opium, phosphoric acid, salicylic acid, santonin, strychnia, stramonium, tar, terebene, and turpentine.

The **Treatment** should be the withdrawal of the drug and the use of astringent lotions, such as those of subacetate of lead, oxide of zinc, or calamine.

ECZEMA.

Eczema (ἐκζέω, to boil over) is a superficial inflammation of the skin, presenting a great variety of lesions, the most common of which is vesication, followed by destruction of the superficial layers of the epidermis, and the prolonged secretion of serum and pus. In this, the typical form of eczema, the eruption begins with some itching or smarting at one spot, which then becomes red, and several minute vesicles form upon it, containing clear yellow serum. They soon rupture, and discharge the serum; and the abraded spots thus produced extend, and coalesce, by the temporary formation, not always very perfect, of fresh vesicles. The secreted fluid is albuminous, and stiffens linen; it is mostly clear yellow, but may be more or less opaque from corpuscular elements. After flowing for a little, it dries up into translucent yellow or opaque whitish or greenish-yellow crusts, which adhere to the surface until detached by accident or lifted by discharge underneath. When they are removed fresh secretion takes place, again drying up into crusts, and this process may go on indefinitely until spontaneous cure or treatment ends it. Sometimes an adherent crust will grow to a great thickness from the secretion underneath. Eczematous patches enlarge by extension at their periphery, which generally shades off into the adjacent skin; and they are often surrounded by other patches, each a quarter to half an inch in diameter, with a small pin-point vesicle, abrasion, or crust in the centre.

Healing takes place by a gradual cessation of the secretion, and covering of the abrasion with sufficient epidermis. This may take place spontaneously under a crust, which may remain long after recovery is advanced. The skin is, however, not normal for some time. Redness and thickening persist, and the epidermis forms large flakes and scales, which are from time to time detached.

The patches of eczema are variable in size, from mere spots to large continuous areas. It occurs in nearly every part of the body; but with especial frequency on the face, ear, scalp, neck, flexure of the elbow, front or back of the forearm, wrist, groin, inner side of the thigh, and flexure of the knee.

Locally, eczema gives rise to severe itching, smarting, or burning, and in certain positions to pain on movement. The general condition of the patient in eczema may be but little affected. In acute cases, with extensive patches, there is some febrile reaction; in many instances the eruption is coincident with, and no doubt induced by, a general malaise, or anæmia, or temporary depression of health; and in prolonged chronic cases the health may be slightly affected as a result of the eczema.

Variations in its course give rise to special names. *Acute* and *chronic* eczema are distinguished by their intensity and duration; but long-continued cases may have frequent acute outbreaks. If the inflammation is intense, with much redness and profuse secretion, it is called *E. rubrum*, or *E. madidans.* In a later stage, or with a less active eruption, the secretion is diminished or absent, and the dermatitis results only in the formation of layers of epidermis, which are successively shed (*E. squamosum*).

E. papulatum is a papular form of eczema, which was formerly described as *lichen.* It is common on the backs of the forearms and on the back.

E. rimosum is the name given to a chronic form in which there is much infiltration and thickening of the skin, with a thin scaly epidermis, only a few scattered discharging points, but several fissures running deeply through the epidermis into the corium, not infrequently bleeding, and excessively sore and tender. This often occurs about the wrists and the lower parts of the forearms.

In other cases the thickening and hypertrophy of the skin form the chief trouble. It is quite rigid and cannot be pinched up. The surface is marked out by small furrows into diamond-shaped areas, and it is white, or powdery from half-detached epidermis. A warty condition may also occur from hypertrophy of the papillæ (*E. verrucosum*).

Ætiology.—Eczema occurs equally in both sexes, and at all ages of life. A number of external irritants, thermic, chemical, or mechanical, give rise to a dermatitis which has all the features of eczema. Exposure to the sun causes so-called *E. solare;* various drugs applied to the skin—*e.g.*, mercurial ointment, and the irritation of very alkaline soaps in some skins—produce an allied condition. In certain trades the hands are constantly irritated, chemically or mechanically, by the substances handled. "Grocer's itch" is an eczematous dermatitis of the hands and wrists from contact with sugar and other groceries. Discharges from the ears, nose, or other parts, and friction of the clothes, may also be causes of the same. In many of these instances the lesion is practically a traumatic dermatitis; and in some of them —for instance, severe sunburn—the inflammation extends deeper into the subcutaneous tissue, and produces larger vesicles or blebs than is common in eczema. They are more deserving of this name when this disease continues after the removal of the cause, or extends beyond the area immediately affected by it. Either suggests the existence of some special tendency to dermatitis on the part of the patient.

Such a tendency to eczema in particular persons does certainly exist, and it is all that can be said about a great number of cases that come under notice. From time to time eczema breaks out on the skin, it is cured by appropriate treatment, and again recurs on slight irritation, or on some slight alteration of general health, or it may be, to all appearance, quite spontaneously. It is only in a comparatively small number of instances that this can be said to be *hereditary*. Amongst the conditions of ill-health to which eczema is in some cases attributable, are various kinds of dyspepsia, intestinal disturbances, whether constipation or diarrhœa, especially in young children, anæmia, the condition of feeble vitality of some children, and in old people, gout.

Anatomy.—In the papillary layer of the corium the vessels are dilated, and there is an increase of fluid and leucocytes in the tissue. Vesicles form in the rete Malpighi or beneath the horny layer. In chronic cases the rete dips down deeply between the papillæ, which are elongated to a proportionate extent.

Diagnosis.—The red, raw surface exuding serum or sero-pus which dries into scabs, is characteristic of eczema, and of more use in diagnosis than the vesicles, which are often of temporary duration. *Scabies* (itch) may be mistaken for eczema; the lesions are mostly scattered pustules or vesicles (even bullæ in some instances), and not continuous patches. It occurs in certain situations, the wrists and fingers in adults, the toes, feet, and genitals in children. The "runs" or burrows, if seen, are conclusive evidence of the itch-acarus. Scabies may set up a secondary eczema. Eczema resembles *sycosis* when it is confined to the hairy parts of the face; the lesions are more superficial than those of sycosis, and not limited to the hair-follicles in the same way: the weeping on removal of crusts comes obviously from intervening skin. *Psoriasis* may be imitated by a dry scaly eczema; patches of psoriasis are more sharply defined, more uniform in shape, round or ringed, covered with thicker, drier scales; and the distribution is characteristic in many cases. *Tinea cir-*

cinata produces circular patches, or rings on the face or elsewhere, which look like a dry eczema; their small number and their circular form should make one suspicious, and the association of ringworm of the scalp, or a microscopic examination, will complete the diagnosis.

Prognosis.—In its acute and subacute forms eczema is amenable to treatment, but many chronic varieties are intractable and last for years. There is a strong tendency to recurrence, which may show itself after long intervals. Only in young children and very old people is it likely that an extensive eczema will help to a fatal termination.

Treatment.—Local treatment is of the first importance in this disease. It is desirable, in the first place, that the eruption should be protected from every sort of irritation. Scratching, alternations of temperature, cold air, washing with plain water, or with alkaline soap and water, will all increase vascularity and secretion, and delay healing. The patient should abstain from scratching, and young children should have the hands tied in gloves, or fixed to the side of the cot. The part should only be washed with thin gruel and water, or with oatmeal and warm water. If there are crusts upon the skin they should be removed so that substances can be applied directly; for this purpose, a bread and water poultice may be left in contact for the night, or for two or three hours, so as to soften the crusts, which may then be carefully removed, or, what is perhaps better, strips of lint soaked in olive oil may be left in contact for three or four hours.

The various local applications that are used serve both to protect from the external air and to modify the condition of the part. Simple oily applications are sufficient, by protection, to promote the healing of some mild cases. But in nearly all cases benefit is derived from the use of sedative and astringent applications. These may be in the form of ointments, lotions, or powders: sometimes one, sometimes another is more suitable. On the whole, ointments are more generally useful, since they can be kept more constantly applied, and lotions must be covered with some impervious material (gutta percha or oiled silk), when the part becomes unduly heated and sodden: otherwise they evaporate, the lint or rag adheres to the part, and considerable irritation is the result. But if there is much secretion on large surfaces, and if the dressings can be constantly looked to, lotions are best. If ointments are used, they should be not merely smeared over the part, but spread upon lint and firmly and uniformly applied, so as to get complete contact; they should, as a rule, be changed twice a day, and the excess of old ointment should be gently removed with a soft cloth, or by washing with oatmeal and water.

The most valuable applications are those of lead: ung. plumb.

acet.; ung. plumbi glyc. subacet.; ung. plumbi subacet. compos. (B.P., 1867, liq. plumbi subacet. ℥vj., camph. gr. lx., white wax ℥viij., oil of almonds ℥xx.); unguentum diachyli of Hebra (made like lead-plaster, with twice the quantity of oil); or solution of subacetate of lead, half a drachm in an ounce of vaseline or lard. Ointments of zinc oxide, oleate of zinc, calamine, and boric acid act similarly. In less acute stages, and especially where pus is secreted, some mercurial ointments may be usefully combined with lead or zinc: such as ung. hydrarg. ammon.; ung. hydrarg. oxidi rubri; ung. hydrarg. nitratis dil. The same substances can be used in lotions, such as the liq. plumbi subacetat. dil.; or a somewhat stronger one, 1 part each of liq. plumbi subacet. and glycerine to 30 parts of water; or lactate of lead made by shaking liq. plumbi subacet. (ʒj. with milk ℥ij.); zinc oxide (ʒss. with glycerine ʒss. to aq. rosæ ℥j.), or calamine (gr. xv.–xx. to ℥j.).

In some cases, especially for use during the day when lotions or ointments may be inconvenient, dusting powders may be employed, particularly oxide of zinc with an equal quantity of starch or French chalk, or boric acid mixed with four or five parts of the same substances. In chronic cases, where there is little discharge and much thickening and scaliness, more stimulant and irritating preparations may be employed, and especially those of tar, creosote, oil of cade, liquor carbonis detergens, and carbolic acid. They may be added to the other lotions or ointments just mentioned—*e.g.*, ung. picis liq. ʒss. or ʒj. to ung. zinci or ung. plumbi ℥j.; or liq. carbonis detergens ʒj. to lot. plumbi or lot. calaminæ ℥vj.; or they may be used alone in inveterate and troublesome cases—*e.g.*, ung. picis liq. ʒss. or liq. carbonis detergens ʒss. to aq. ℥vj. Ichthyol also gives good results as an ointment (ʒj. to vaseline ℥j.), or as a lotion (ʒj. to ℥vj.).

Most of these methods of treatment will relieve the itching which accompanies the disease. Stronger sedatives are sometimes employed—for instance, cocain; but the exposed surfaces render such applications very unsafe.

The *internal* treatment of eczema must be directed to correcting every fault in the digestion or general health that may be detected. Gastro-intestinal troubles of children, and dyspepsia in others must be treated by suitable remedies; delicate and anæmic patients should take iron, cod-liver oil, quinine, &c. If gout is a fact in the patient's history, laxative salines should be given; but it is not right to prescribe for gout unless there are some other indications than the eczema itself. Internal treatment may be called for to allay the terrible itching of some cases; for this purpose chloral and potassium bromide are most useful; the former especially in children—for instance, 2 or 3 grains in syrup to a child of six months given at bedtime and repeated near midnight; and the bromide in older people. Hyoscyamus, or tincture of hops, may be also given, and quinine seems to allay itching in eczema as well as in chronic urticaria.

Of specifics for eczema there are few. Arsenic is undoubtedly of great value in many cases, but not in all. It is most suited to chronic, frequently recurring cases, and least so to acute eruptions. Recurrences are often checked at once by its exhibition. It should be given after meals in small doses gradually increased. There is no other remedy that has gained much reputation as a specific in eczema. Crocker has seen good results from spirits of turpentine, given in doses of 10 to 30 minims in mucilage, three times daily after meals, the last dose not later than 6.30 p.m.; water, or barley water, must be drunk freely during the time. He has also had success in inveterate cases with another indirect method of treatment—namely, counter-irritation over the neck or loins with mustard or blistering fluid, in order to influence the vasomotor system. Malcolm Morris recommends antimony in small doses.

In acute eczema the diet should be that of a febrile case, chiefly milk and farinaceous food. In chronic cases very little alteration is required. Stimulants should be used in moderation only; and salt meats, as likely to increase thirst, may be well avoided. The patient should of course, abstain from any food that obviously disturbs digestion.

The treatment of eczema in particular localities may be briefly mentioned.

Scalp.—Cut the hair short, remove crusts by oil or poultices; apply weak iodoform ointment in pustular cases, or a mixed ointment of zinc oxide, lead acetate, and dilute mercuric nitrate ointment (ung. metallorum). In drier eczema, mixed with seborrhœa, use tarry applications—*e.g.*, liq. carbonis detergens.

Hairy parts of the face.—Cut the hair and shave as soon as it can be borne, then use astringent ointments; extract the hairs if pustules form around them.

Lips.—Mild astringent ointments of lead, zinc, or yellow oxide of mercury. Crocker recommends a formula by Hebra—viz., acid. carbol. ʒij.; glycerini, ætheris, aa. ℥j.; sp. vini rect. ℥vj.

Palms.—After removal of crusts, apply the ointment spread upon strips of lint separately to each finger, and fit a kid glove over all. In chronic cases remove the thickened epidermis by the use of salicylic acid plaster or by soaking with pancreatic emulsion (Crocker), or papain (M. Morris). Salicylic acid or mercurial ointments may then be applied.

Nails.—Eczema of the nails is rare; they have a dirty, yellowish colour, and are pitted, grooved longitudinally, thickened, thinned, or split. Apply mercurial ointments, or those of salicylic acid, or of tar, wrapping up the ends of the fingers completely.

Legs.—This often results from varicose veins. Keep the patient in the recumbent position and raise the legs; or bandage carefully from foot to knee, or use Martin's rubber bandage. Apply astringent ointments or lotions.

IMPETIGO.

This name was formerly applied to many pustular eruptions, but it is now almost limited to one on the scalp, and to an eruption which has been separately described as *Impetigo contagiosa.* What is considered distinctive of I. contagiosa is the occurrence of flat vesicles, enlarging into pustules from a quarter to half an inch in diameter, with scarcely any surrounding inflammation. They occur especially on the face, about the mouth, nose, and chin, and in the occipital region. They are often few in number and discrete; and they appear to cause similar lesions by contact in other parts of the skin, or in other persons. It is admitted that they often coexist with pediculi capitis, which are the commonest cause of *Impetigo capitis* of children, and whether there is any cause of contagion in the pus more than is contained in pus from other sources, remains to be be seen. Pyogenic organisms, especially streptococci, are present.

Impetigo of the scalp forms thick crusts of yellowish, or greenish-yellow colour, matting the hairs together; it is most marked in the occipital region when due to pediculi. The suboccipital glands are generally enlarged and may suppurate.

Treatment.—In all cases pediculi should be sought for; their occurrence, past or present, is often shown by the nits adherent to the hair (*see* Phtheiriasis). Apart from the cure of pediculi, the local lesions of impetigo may be treated by removal of the scabs and the use of a dilute white precipitate ointment (1 to 4), or the mixed ointments of zinc, lead, and mercury. Tonics, such as cod-liver oil and iron wine, are often desirable at the same time.

ECTHYMA.

Ecthyma is a somewhat similar suppurative lesion, consisting of scattered pustules with resulting scabs on the trunk and extremities, and occurring in cachectic and anæmic persons. The lesion is deeper-seated than impetigo and extends to the corium, and there is more red areola around the pustule and scab. Pyogenic organisms are found in them. The treatment is similar.

LICHEN.

This term has been long in use to signify any sort of papular eruption, and on this view a number of species used to be described which are now classed with other diseases. The name is reserved for a quite small number of complaints.

LICHEN SCROFULOSUS.

An eruption consisting of small papules, at first red or pink, later fawn-coloured or almost yellow, arranged in roundish groups, or circles, or segments of circles. On the older papules a minute scab is formed, and after a time the papules subside and leave only a yellowish pigmentation. They occur on the trunk, especially at the sides, and rarely on the limbs; the occurrence on the limbs is more frequent in children than in adults. Itching is absent, or very slight. The disease progresses by the appearance of fresh crops of papules from time to time, so that it may last for months or years.

The chief factor in its **Ætiology** seems to be the condition of health known as scrofulous or strumous; many patients have enlarged lymphatic glands, caries, or other bone lesions, or ulceration of the skin; but phthisis is not common. It occurs in both sexes, but has been seen more often in males. It is most common in children, and rare after early adult age.

According to Kaposi the lichen papule is formed by an infiltration of cells in the papillæ round the follicle, and the central scale by a collection of epidermis at its dilated orifice.

The **Treatment** consists of the use of cod-liver oil internally; and externally, either cod-liver oil (Hebra) or some other emollient, such as vaseline, or vaseline with liq. plumb. subacet., thymol, or oil of cade (Crocker).

LICHEN RUBER.

This is known in two forms—*Lichen ruber planus* and *Lichen ruber acuminatus*, of which the former is more common in England, and the latter appears to be more often seen in Vienna.

L. planus consists of raised flat patches of a dull red colour, and a surface smooth, shining or covered with quite small scales. The patches arise from the aggregation of papules, which are at first discrete, and then become continuous by the growth of fresh papules in the intervening spaces. The papules are flat, shining and angular, sometimes with a minute depression in the centre. The eruption is more or less symmetrical, and appears first on the wrists and forearms, and on the inner side of the knees, and then on the extensor surface of the arms or legs, the ankle, foot, the flank, hip, and lower part of the abdomen. It also tends to appear on parts subject to pressure—for instance, the waist, the legs where the garter presses, and the palms and soles: but in these last two situations there is only a general thickening of the epidermis, with white spots where the horny layer is cracking. The rash on the skin is often associated with white spots on the tongue and inner side of the cheeks. The head and face are said never to be affected. There is only moderate itching, and the health is influenced simply in proportion to the extent of skin involved. In generalised extensive disease marasmus and death may ensue. When the lesions subside they leave a very persistent stain.

L. acuminatus begins with conical red papules, capped with scabs, which are at first widely separated; but as more and more of the intervening skin is affected, continuous patches are formed. It may develop rapidly over the whole of the body, or begin in the flexures alone. Eventually the whole skin may become reddened, scaly, thickened, and infiltrated: the skin of the palms, soles, fingers, and toes is deeply fissured, and the nails are rough, thickened and broken, or thin and brittle.

Ætiology.—In many cases no cause can be discovered; in others worry, anxiety, insufficiency of food, &c., have preceded the disease. It is most common between the ages of twenty and fifty, and rarely attacks children. English cases have been more common in men, and the Vienna cases in women.

Pathology.—The inflammatory process in L. planus begins round a sweat duct in the upper part of the corium, and this is followed by thickening of the rete Malpighi and its extension downwards between the papillæ. In the acuminate form the hair-follicles are the centre of inflammation.

Treatment.—The treatment is not unlike that of psoriasis. Internally arsenic should be given steadily in full or increasing doses for a considerable time; it is most successful in chronic cases, and less certain in acute. The local treatment consists in the use of tarry preparations, such as ung. picis liq., ung. creosoti, liq. carbonis detergens, thymol, and carbolic acid. If there is much hyperæmia more soothing applications, such as lead or zinc lotions, may be desirable for a time. The general health also requires attention; bodily and mental rest, nutritious food, the usual tonics, and, perhaps, change of air.

OTHER SKIN DISEASES DESCRIBED AS LICHEN.

L. simplex and *L. Agrius* are papular forms of eczema; *L. lividus* is purpura round the hair-follicles; *L. urticatus* is urticaria in children; *L. circumscriptus vel circinatus* is now recognised as a *Seborrhœa; L. pilaris* is now called keratosis pilaris; but Crocker describes under the title *Lichen pilaris* a very rare disease, the *Lichen spinulosus* of Devergie, an inflammatory disease of the hair-follicles, in which a spiny epidermic peg occupies the centre of the papule. It is to be treated by alkaline baths, and subsequent friction with soap liniment.

PRURIGO.

This is a papular disease accompanied with severe itching (*prurio*, I itch). It must be distinguished from *pruritus*, which means simply the sensation of itching, and not structural change. Thus, we have pruritus in prurigo itself, but also in eczema, in jaundice, and in other conditions. One disease that is often described as prurigo (*P. senilis*, *P. pedicularis*, *Phtheiriasis*) is obviously the result of the irritation of the body-louse, and I shall speak of it when treating of parasites. Prurigo consists originally of minute papules in the skin, which are at first not so much visible as palpable, having the colour of the skin, and only later becoming pink and red. They are not collected in groups but scattered. As they are accompanied by severe itching, they are soon scratched, the heads of the papules are removed, and a small blood-scab is the result. More violent scratching leads to enlargement of the papillæ, thickening and roughening of the skin, the natural furrows are deepened, the surface is covered with mealy scales, the downy hair is destroyed, and when the hand is passed over the skin it feels like a nail-brush or like rough brown paper. Besides the excoriation of the papillæ, more extensive scratch-marks, abrasions, and scars may be caused, and other secondary lesions, such as eczematous patches, urticaria, pustules, enlargement of the femoral, axillary, or elbow glands, and finally, more or less deep pigmentation of the skin.

The parts first affected are the extensor surfaces of the legs and arms, especially the former. The chest, back and front, the abdomen, and gluteal regions are all affected, even a few papules may appear on the face; but the flexures of the elbow and knee, the axillæ, the genitals, the ankles, wrists, palms, and soles are always spared.

Ætiology.—It is more common in males than in females, and among the poorer classes. Different statements are made as to the influence of cold, but it is certain that winter cold does cause in some people a pruriginous condition, especially of the legs (*P. hyemalis*). It commonly begins in infancy, and continues, unless vigorously treated, for the rest of life.

Varieties.—*P. mitis* and *P. ferox* have been described: they appear to differ only in intensity. Hebra regarded as a special and incurable form the very intense cases which he saw in Vienna; but there seem to be cases intermediate between those and the milder forms commonly seen in England.

Pathology.—The early change is an exudation into and above the papilla of leucocytes or serum, which also infiltrate, and lift up the epidermis. As to its origin beyond this, little is known.

minuria. If recovery takes place, there may be a relapse; but the disease is often persistent, and death occurs from marasmus, diarrhœa, pneumonia, or bronchitis.

It occurs in both sexes and at all ages, but is comparatively rare in children.

Anatomy.—According to Crocker, it is dermatitis, at first superficial, but afterwards involving the whole depth of the skin, and resulting in new connective tissue, cicatricial contraction of the same, pigmentation, hyperplasia of elastic fibre bundles, and obliteration of the papillæ, and of the sudariparous and sebaceous glands.

Diagnosis.—Pityriasis rubra has certain points of similarity with eczema, psoriasis, and pemphigus foliaceus. From the first it is distinguished by the extent of body involved, by the absence of secretion, and by papery scales instead of yellow crusts; from *psoriasis* by the extent, by the absence of infiltration, and by the scales not being massed into thick flakes; from *pemphigus foliaceus* by the absence of bullæ with discharges preceding the scales. From *lichen ruber*, also, it is distinguished by the absence of papules and of infiltration. Some of the forms of *dermatitis* mentioned below (p. 936) may resemble it closely.

Treatment.—Emollient applications are mostly recommended, such as olive oil, linimentum calcis with zinc oxide and calamine, lead and zinc ointments, glycerine of lead subacetate, or lactate of lead; or weak tarry preparations, such as carbolic oil, or liq. carbonis detergens in vaseline (1 in 8, Pye-Smith), or ichthyol soap. But stronger tar preparations may be too irritating. The applications must be frequently made over a long period. Internally, tonics and nutritious diet must be given, and in the older cases arsenic is of value.

OTHER SKIN DISEASES CALLED PITYRIASIS.

P. capitis is a form of *seborrhœa; P. versicolor* is due to the growth of a fungus, and is now called *Tinea versicolor*.

PSORIASIS.

This disease consists in the formation of raised red patches, covered with thick, silvery-white adherent scales. In a great number of instances the lesions appear first on the knee over the patella, ligamentum patellæ and tubercle of the tibia, and on the elbow over the olecranon. It begins with papules, which enlarge into round flat plaques; quite early the papule is seen to be covered with an opaque scale, and with its enlargement in size, the scale becomes thicker, especially in the centre, and silvery-white in appearance. The scale is rather firmly adherent, and coextensive with the red plaque, so that the red colour can often only be seen at the edge. If the scale is removed it leaves a shining, moist-looking, but actually dry bright red surface, in which examination with a lens will show a number of deeper red points, the hyperæmic papillæ. The patches are at first roundish or circular, and enlarge to half an inch, an inch, or more in diameter; fresh patches come out near the first or in other parts of the body. If a patch becomes very large it may heal in the centre, and thus form a ring; coalescence with other rings will produce serpiginous or gyrate figures. The patches may spread sufficiently to cover large areas of the body continuously, so that the original shape of the spot cannot be detected. The old names given to indicate these different stages have little more than a descriptive value, such as *P. punctata*, *P. guttata*, *P. circinata*, *P. gyrata*, *P. diffusa*, *P. universalis*.

Next to the knees and elbows, the adjacent extensor surfaces of the leg and forearm are most commonly affected, and then the thighs, back, loins, chest, and abdomen; and in all regions a very striking symmetry is observed. The face and scalp are not often attacked, and the palms and soles rarely. The nails are not infrequently involved: they become variously altered, opaque, thickened, pitted, furrowed transversely or immensely thickened, and discoloured.

The amount of scale varies in different instances or in the same case at different times. In *P. rupioides* the scales are heaped into small conical masses, each on its circular base. The eruption is always dry, never moist or scabbing as in eczema. Itching is variable, but not, as a rule, severe. The general health is often perfectly good, or even robust.

The disease breaks out spontaneously, often in early childhood, and even if not treated subsides after three or four months, to recur again after a quite uncertain interval. Sometimes the recurrence is twice a year ("spring and fall" as it is often expressed), or a period of years may intervene. In other cases a slight amount of eruption persists, and extensions take place from time to time. During recovery, pigment stains mark the situation of the patches, especially after the use of arsenic.

Ætiology.—It affects both sexes, and nearly all ages; but it more commonly begins in early life. The only other certain fact in its causation is that it is hereditary; the view that gout and scrofula are causative antecedents has little in favour of it.

Pathology.—It is an inflammation of the papillæ and corium with increase of the rete, downgrowth of the same between the papillæ, which appear correspondingly enlarged, and increase of the horny layers.

Diagnosis.—In most cases it cannot be mistaken. Patches of dry *eczema* may resemble it, but the edges are not so sharp and the scales are not so thick and silvery. In psoriasis of the scalp the scales are often yellow, and look like impetigo crusts; but psoriasis spreads beyond the scalp on to the forehead or neck, and there is always psoriasis of some other part of the body which will be distinctive. These last two points will also serve as between psoriasis and *seborrhœa.* The features of lichen ruber and pityriasis rubra have been sufficiently described. Patches of *tinea circinata* may look like psoriasis, but their small number, want of symmetry, small scales, and the results of microscopic examination will show their nature. *Lupus erythematosus* is recognised by its position on the face, the greater thickening of the skin, the sebaceous plugs, and the scars; and *scaly syphilides,* by the small size of the lesion, the slight scaliness, the browner colour, and the concomitant symptoms.

Treatment.—The best treatment is a combination of internal and external remedies, but one of these alone will suffice in some cases. *Internally,* arsenic is most frequently successful; it may be given in the usual way—that is, increasing doses of liquor arsenicalis, beginning with 3 or 5 minims, and stopping at 10 or 15 minims, always after meals. Other forms in corresponding doses, arsenious acid in pills, liq. arsen. hydrochl., or liq. sod. arseniatis, may be equally well employed. Some other drugs are said to be also of use; carbolic acid in ½-grain doses (Kaposi); turpentine in 10 to 30 minim doses (Crocker), and iodide of potassium. The last requires to be given in very large quantities, and cannot always be borne.

The best *external* treatment is that by tar and allied preparations: ung. picis liquidæ, creosote ointment, ointment of cade oil (ʒj. to ʒiv. to ℥j.), or liq. carbonis detergens (ʒij. to ℥j.). The preparation should be both rubbed into the part and left in contact, the limbs being enveloped in old flannels to prevent staining of the clothes. If the scales are very abundant it may be necessary to remove them first by the use of alkaline baths, the wet pack, soft soap, or vaseline. Besides the tarry preparations mentioned above some others are efficacious: chrysarobin ointment, which must be used with caution, as it often sets up a dermatitis beyond the limits of its application, and stains the hair and clothes of an orange or golden colour; pyrogallic acid (ʒss. or ʒj. to ℥j. of benzoated lard), which must be used over a small area at a time. Crocker also recommends turpentine or oleum pini sylvestris, alone or diluted with olive oil. Thyroid extract was first used for psoriasis by Bramwell, who observed that in patients with myxœdema under treatment by this extract, the skin desquamated. Sometimes good results have followed, at others the method has failed.

It has been stated that many patients are in robust health; if this is not the case, any defect, such as anæmia or strumous delicacy, should be met by iron, cod-liver oil, or quinine, which need not interfere with the specific arsenical course.

ANOMALOUS FORMS OF DERMATITIS.

Besides those due to the internal administration of drugs, other cases of more or less diffuse inflammation of the skin occur, which it may be difficult to class with any of the preceding forms of disease, though their predominant features may be those of one or the other. They may be provisionally called dermatitis. Such, for instance, are the eruptions produced by the irritation of some plants, especially those of the order *Anacardiaceæ*, to which Rhus toxicodendron, and the Indian marking-nut belong. Another form of dermatitis is one that is occasionally seen in chronic Bright's disease towards the end of the illness (p. 795). It often begins as papules or larger elevated patches of red inflamed skin, which ultimately coalesce, so that the whole body may be covered with red thickened skin. Subsequently desquamation takes place, and some cases have a close resemblance to pityriasis rubra. One may consider also under the head of dermatitis the cases of epidemic skin disease recently recorded (Savill), of which some were more like eczema, others more like pityriasis rubra.

It will be observed how frequently dermatitis is due to some kind of poison, either given as a drug, or arising from decomposition of food (ptomaïnes), or from septic processes, or from retained excretory compounds.

HYPERTROPHIES OF THE SKIN.

CALLOSITIES AND CORNS.

These are produced by friction and pressure.

A *callosity* consists of a hypertrophy of the horny layers of the epidermis, and is familiar on the ball of the great toe, the heel, the hands of the working man, of oarsmen and others, the tips of the fingers of those who play the violin, &c. Callosities of apparently spontaneous origin have also been seen occasionally on the palm of the hand (*Tylosis palmæ manus*, Hebra).

The *corn* (clavus) is a local thickening of the epidermis resulting in a conical downgrowth, which presses upon the subjacent papillæ, causes their atrophy, and sets up inflammation and hypertrophy in the surrounding papillæ. Corns are common, as is well known, on the toes, especially the outer side of the little toe, the dorsum and the sides of the other toes. The pain of the ordinary corn is largely due to the little plug being driven down on the cutis beneath, but spontaneous shooting pain is often present. When the corn lies between two toes and is kept constantly moist, the thickening is less marked; but the inflammation is more obvious, and the part is often extremely tender (*soft corn*). Occasionally corns will inflame and ulcerate, or a cyst or bursa forms under the corn, constituting a *bunion*.

Treatment.— Corns may be cured, and almost entirely prevented, by the use of properly shaped boots. The sole should be as large as, or slightly larger than, the sole of the foot as it shapes itself in the standing position with the weight of the body upon it. If the boot sole is narrower than the sole of the foot, the upper leather must be in tight contact with the edge of the foot in any movement, and constant friction is the result. The inner edges of the sole should be straight, and pointed boots should be strictly avoided. If corns have formed, they must be treated by soaking in hot water, and shaving with a sharp knife or razor, when the dry white plug will be met with and can be removed. A corn plaster may then be worn, or the toe may be simply strapped with a good linen plaster like Leslie's, by which, with properly constructed boots, the friction will be reduced to a minimum. Soft corns may also be carefully shaved, and pressure removed by cotton wool between the toes, or by a turn or two of narrow strapping below the corn. Salicylic acid in a two per cent. ointment, or salicylic acid plaster, is effectual in removing the thickened epidermis, and the tender foot may be benefited by the use of alum or tannic acid lotions. But in all cases a sufficiently broad-toed boot, with a wide sole and a low heel, is the one requirement for permanent relief.

ICHTHYOSIS.

In this disease the skin is dry and rough from thickening of the epidermis. It is congenital, though it is not seen until some weeks or months after birth, and it occasionally runs in families. In its mildest form (*Xerodermia*, dry skin) the skin is rough, dry, and dirty looking, especially over the extensor surfaces of the legs and arms. In more pronounced forms (*Ichthyosis simplex*) the whole of the body is more or less affected, the limbs most, the scalp, face, palms, soles, genital organs, and flexures of the joints least. The skin is not reddened; but it is covered with thin epidermic scales, whose shape is more or less determined by the folds on the skin, and on the extremities inclines to a polygonal or diamond shape; and along these lines the scale is partly detached, while within them it is adherent. Still, a certain amount of shedding is constantly taking place. There is an absence of perspiration, but the sebaceous secretion is mixed with the epidermic scales, and with the adherent dirt contributes to give a grey, or greenish dirty appearance to the whole of the skin, or its most affected parts. The health is not affected by it, but the growth of the patient is sometimes stunted; and eczema is a common occurrence.

There is a gradation between this and *Ichthyosis hystrix* (porcupine skin); but the condition known under this name is often localised, or unilateral, and in some cases follows the track of cutaneous nerves. It consists of thick green or greenish-black plates or masses of hypertrophied epidermis, of square or polygonal shape, rising a quarter or a third of an inch above the skin, closely fitting together, like a mosaic pattern. Under the epidermic masses the papillæ are hypertrophied. Microscopic examination shows that in all forms the accumulated masses consist of aggregated epidermic scales. In Ichthyosis hystrix the papillæ are hypertrophied, and the horny layers of the epidermis dip down into the inter-papillary spaces. In Ichthyosis simplex it would appear that the cutis is unaffected.

Treatment.—Complete cure cannot be effected, but considerable relief can be obtained. The scales should first be removed by hot baths, alkaline baths, friction with soap, &c., and then some emollient application should be kept constantly applied, such as glycerine of starch, vaseline, cold cream, olive oil, &c. On the cessation of the treatment the former condition will return. For the smaller growths of Ichthyosis hystrix, Crocker recommends removing the horny caps, and painting the base with a saturated solution of salicylic acid in alcohol.

KERATOSIS PILARIS.

This consists of small papules, the size of a pin's head, which occur mostly on the extensor surfaces of the limbs, and are formed by accumulations of epidermis at the mouths of the hair follicles. The hair may pierce the centre, or more often it is coiled up in the centre and broken off. The papules are often brown or black from adherent dirt.

The **Treatment** is similar to that of ichthyosis.

WART.

(*Verruca*).

Warts are small excrescences from the skin, consisting of hypertrophied papillæ capped with horny epidermis. They may be flat, hemispherical, or pointed: and the larger may be lobulated, or digitate. They are generally pale pink, or yellowish, or pale brown in colour. They occur especially on the backs of the hands, and are commonest in children and young people. They often disappear spontaneously after a long time.

Verruca acuminata (*Condyloma*) occurs on the perineum, on the glans penis, or labia, about the anus, mouth, and other moist situations. Condylomata are generally pink or red, pointed or club-shaped, or variously modified in shape by mutual pressure, and in moist situations secrete a whitish puriform fluid. They occur as the result of irritating discharges, like those of gonorrhœa or soft sores, or as the result of friction.

Treatment.—Warts are commonly treated by the application of nitrate of silver, glacial acetic acid, saturated solution of chromic acid, or other caustic. Saturated solution of salicylic acid in alcohol frequently applied is also effectual. Thorough cleanliness and astringent lotions may suffice for the acuminate forms. Repeated doses of sulphate of magnesium, 2 or 3 grains for children, or 30 grains for adults, three times a day, are said by French physicians to cure warts, and this is confirmed by Crocker.

Verruca necrogenica is a lesion due to inoculation, sometimes certainly tubercular, by blood or fluids from the dead body (p. 453).

CORNU CUTANEUM.

Horny growths, sometimes several inches in length, and generally twisted or bent, have in rare cases been seen. They are, as a rule, solitary. They consist of accumulated epidermic layers on a base of hypertrophied papillæ. The **Treatment** consists of removal and cauterisation of the base.

SCLERODERMIA.

Sclerodermia (hard skin) may be general or circumscribed. These were originally described as separate lesions, but it was shown by Fagge that they are essentially the same, and Crocker has proved the identity of their anatomical changes.

In *diffused sclerodermia* there is a general hardening or induration of the skin, which begins most commonly about the face, neck, shoulders, chest, and arms, and may gradually extend to the lower part of the body. There is at first no change in colour, but the skin is hard, rigid, inelastic, and cannot be pinched up into folds. As it goes on, the movements of the limbs are hindered, the joints are more or less fixed, the chest is limited in its respiratory movements, and if the face is affected it loses its power of expression; the mouth can be opened with difficulty, but the eyelids often retain their mobility. Subsequently, the skin becomes shiny and glossy, irregular patches of pigment appear, and here and there are areas of vascular dilatation giving a pink or violet colour. The secretions of sweat and sebum are diminished. The course of the disease is slow, and it extends over years, eventually, in many cases, subsiding entirely. During this time the patient's health is practically unaffected, but rheumatism and cardiac troubles have been noted as occasional complications. In the skin itself eczema, erythema, and ulceration may occur.

In some cases, the disease begins with more thickening or œdema of the skin, and this, according to Crocker, tends to result in an atrophied, rigid, tight condition, which is much less liable to spontaneous recovery than the simply indurated forms.

The disease occurs in young adults and middle-aged persons, less frequently in children, and hitherto not under the age of thirteen months (*see* Sclerema Neonatorum). It is more frequent in women than in men, but little is known of its causes.

In *circumscribed sclerodermia*, or *morphœa*, there is an unsymmetrical patch of two, three, or more inches in diameter, frequently corresponding to the distribution of a nerve. For instance, a patch may occur over the distribution of the supraorbital nerve on the forehead; the trunk near the breast, and the limbs, are also common places for the eruption. The patches are irregular in shape, or may be in the form of bands, round or along a limb. They are of a dead-white ivory colour, surrounded by a violet or pink zone of dilated vessels. The skin is smooth and dry, and may often be pinched up; it may be level with the healthy skin, or below or above it. The disease lasts several years, and then subsides and disappears, or it may extend into the diffused form, or persist in an atrophic condition. Circumscribed sclerodermia is also more common in women than men, and can sometimes be referred to local irritation as a cause.

Anatomy.—The epidermis is unaffected except for some pigment in the rete; there is a considerable overgrowth of connective tissue in the corium and subcutaneous tissues; the deeper vessels are surrounded by numerous leucocytes; and the superficial vessels are often contracted and empty. Leucocytes also surround and may obstruct the sweat-gland ducts, and the muscular fibres of the skin are hypertrophied.

Treatment.—This is not very satisfactory. The patient should be kept warm at all times, and tonic remedies should be given. Locally, emollient applications and friction, and shampooing to restore the circulation in the skin may be tried. Galvanism has also been used. Thyroid extract has been tried without result.

SCLEREMA NEONATORUM.

This is a peculiar induration of the skin, which is either congenital or begins shortly after birth in feeble infants with deficient circulation. It may begin in the lower extremities and spread to the rest of the body, or it occurs in scattered patches on the thighs, buttocks, trunk, arms, and cheeks. The affected parts feel quite hard and firm, suggesting that the subcutaneous tissue has been frozen. The patches have a well-defined edge, are slightly raised above the surface, and sometimes have a bluish-red colour. They only pit after very prolonged pressure. The children are cold and drowsy, with small pulse and feeble respiration. They often die from collapse or diarrhœa, but occasionally recover. The cause of the change is not well understood. It may be confounded with a true *œdema*, which pits readily on pressure (Crocker).

Treatment.—The child should be kept warm and efficiently fed, by a nasal tube, if necessary.

ATROPHIC CONDITIONS OF THE SKIN.

Besides senile atrophy, in which the skin becomes dry, inelastic, wrinkled, and often pigmented, the following conditions may be described as atrophy of the skin:—

ATROPHODERMIA NEURITICA.

This, the "glossy skin" of Paget, follows upon neuritis and other lesions of the nervous system. It is especially well seen in the fingers, of which the skin becomes smooth, shining, dry, the colour pink or red, the whole finger tapering, and the nails curved longitudinally and transversely. With this is a severe and persistent burning pain.

STRIÆ ATROPHICÆ.

These are the translucent, scar-like lines which form in parts of the body which have undergone considerable distension, such as the abdomen after pregnancy (*lineæ gravidarum*, *lineæ albicantes*), the breasts after lactation, the abdomen, thighs, legs, and arms after extreme anasarra, and the shoulders, breasts, and thighs from obesity, or the presence of more localised fatty tumours. A similar change may occur in the skin without any preceding distension, coming on spontaneously and at first without the knowledge of the patient. It is then seen mostly about the buttocks, thighs, knees, and ankles, and may be in the form of lines or spots. In all these cases the skin is really atrophied, the papillæ are smaller or absent, the epidermis thinned, and the subcutaneous tissue and glands atrophied. But an early vascular, or even hypertrophic, condition has been observed in idiopathic cases.

ATROPHODERMIA PIGMENTOSA.

(*Xerodermia of Kaposi.*)

This is a remarkable and rare disease, which consists of combined atrophy of the skin, increased pigmentation, and dilatation of the vessels. It occurs equally in males and females, and has a tendency to affect members of the same family, without being actually hereditary. It begins in childhood, with pigment spots, or with erythematous spots, which soon fade into pigment. These form over the face, neck, scalp in the temporal region, outer side of the arm and forearm, and back of the hand. The pigment spots afterwards become atrophic, and patches of white, depressed, shrunken skin form among them. These white spots are slightly contracted, and difficult to pinch up; and subsequently sufficient tightening of the skin may occur to depress the eyelids, and set up conjunctivitis. On the atrophic area there occur pink spots of dilated vessels, which gradually enlarge. The disease may remain stationary for a long time, and never spread to other parts of the body; but eventually warty growths develop out of either the dilated vessels or the pigment spots, and these subsequently grow into tumours of an epitheliomatous nature. These fungate, discharge or bleed, and other tumours forming in remote parts of the body, the patient is carried off by exhaustion. No treatment is of any avail.

Kaposi describes a *Xeroderma albidum* (atrophoderma albidum, Crocker) affecting the leg from the thigh downwards, and sometimes the arm down to the hand, in which the skin is atrophied, and then stretched. It begins in early childhood and remains stationary.

ALTERATIONS OF PIGMENT.

Increase of pigmentation arises under circumstances in which hyperæmia is induced for a longer or shorter time. The most familiar is exposure to the sun or to the wind; but in the foregoing sections it will have been noticed how frequently pigmentation is said to follow upon the different forms of dermatitis —for instance, eczema, erythema, pemphigus, lichen, and psoriasis; to these may be added erysipelas, syphilitic eruptions and ulcerations, especially old-standing ulcers from varicose veins in the lower extremities. The application of blisters and mustard plasters is often also followed by staining, a fact which should make one careful how one orders these counter-irritants to the neck or arms of young ladies. Another common traumatic cause of increased pigmentation is the scratching which is indulged in to relieve pruritus, especially that of severe prurigo, whether idiopathic or from the presence of pediculi.

Some disorders of the skin in which hyperæmia is not a marked feature are also accompanied with pigmentation, such as sclerodermia, Kaposi's xerodermia, and leucodermia, which will be described presently. As a result of internal disease, we see pigmentation of an extreme form in Addison's disease, to a less extent in some cases of lymphadenoma, in the cancerous cachexia, in malaria, and in some cases of tuberculosis. The possibly nervous origin of this has been alluded to under Addison's disease. Except in this last case (melasma suprarenale) it is not common to employ any special name, but the term *melanodermia* and *chloasma* (from χλοάζω, to be pale green) have been used, of which the former is decidedly preferable. In all the cases which are due to a removable cause, the pigmentation will, in its absence, eventually disappear; on the other hand, it persists in incurable cases like Addison's disease, and increased pigmentation coming on in old age does not, of course, undergo any improvement. Local collections of pigment occur, as pigment moles, and pigmented warts. The special forms to be here described are lentigo or ephelis, and chloasma uterinum.

Deficiencies of pigmentation are seen in albinism and leucodermia.

LENTIGO.

(*Ephelis. Freckles.*)

Yellow, orange, or yellowish-brown maculæ appear on the face, neck, forearms, and backs of the hands, from exposure to the sun under certain conditions. They are most marked during the summer-time, and fade or entirely disappear during the winter; they are first seen about the age of late childhood, and rarely in advanced life, and they affect especially people with fair hair and blue eyes (xanthochroic type). Of a similar kind are the mottled patches of brownish-yellow pigmentation which are seen on the fronts of the legs and the backs of the forearms of those who are in the habit of sitting close to a large fire—*ephelis ab igne.*

CHLOASMA UTERINUM.

The pregnant state, as is well known, is commonly accompanied by an increased pigmentation of the nipples, axillæ, and the line between the umbilicus and the pubes. In some women a curious band of pigmentation forms, under these circumstances, on each side of the forehead, just below, but not touching, the margin of the scalp. It is narrow in the middle line, widens out as it reaches the temple, and may extend over the zygoma on to the cheek; it is continuous, or broken into separate small patches. The colour is yellow or brown. With it may be associated the familiar dark ring round the eyes. This frontal chloasma sometimes recurs with each successive pregnancy, and disappears with delivery. It may be due to other uterine disturbances—*e.g.*, dysmenorrhœa—and sometimes no cause can be traced.

Treatment.—Corrosive sublimate has been most used as a local application, in a solution of the strength of one or two grains to an ounce of almond emulsion; and also solution of citric acid, carbolic acid, and other mild caustics, by which the epidermis is removed, and with it the pigment. But it tends to recur. Crocker hopes better results from salicylic acid paste, or plaster, or a saturated solution of the acid in alcohol kept on for some hours.

ALBINISM.

This is a congenital deficiency of colour, not only in the skin, but also in the hair and in the iris and choroid. It is at once recognised by the white hair and the pink eyes; and there is commonly intolerance of light, from the want of pigment in the latter. It occurs in dark races as well as in the pale-faced; and in various animals—cats, mice, and others.

LEUCODERMIA.

Scattered white patches, occurring in any part of the body—the neck, chest, abdomen, arms, or legs—are known as leucodermia. The patches vary from half an inch to several inches in diameter, are irregular in shape, but have convex borders, are frequently grouped together, and, gradually enlarging into one another, may form convexed scalloped borders. Around the margin of each patch the skin becomes darker than normal, and the colour gradually fades as it is farther and farther from the white patch, until the normal tint is reached, some half-inch or so away from it. There is thus in leucodermia a double process: a deficiency of pigment over a certain area, and an increase of pigment around it. Beyond these alterations of the pigment, the skin is quite unchanged; but hair growing from leucodermic patches also loses its colour. The disease is an acquired one; it is more common in hot climates, and among dark races, than in England. Its association with certain diseases has been rather frequently noted—viz., morphœa, alopecia areata, Addison's disease, and exophthalmic goître; but its cause is otherwise entirely unknown. Improvement may take place spontaneously; but treatment is of little avail. Unsuccessful attempts have been made to develop pigment on the white patches by blisters, ammonia, or other irritants. Staining with walnut-juice may be used to mask it temporarily.

NEW GROWTHS IN THE SKIN.

Under this head are included not only definite tumours, but also certain infiltrations with granulation tissue (*granulomata*) determined for the most part by some infecting virus or micro-organism: such are lupus, leprosy, tubercle, rhinoscleroma, and frambœsia. Syphilides, which belong to this group, have been already described (pp. 126, 128). For vascular tumours, or *nævi*, *carcinoma*, *epithelioma*, *rodent ulcer*, and *sarcoma*, the reader is referred to works on Surgery.

LUPUS.

Lupus is a disease of the skin consisting of a new growth in the cutis vera, which terminates in cicatrisation, with or without preceding ulceration. Two forms occur, which differ in important particulars. They are *Lupus vulgaris* and *Lupus erythematosus*.

LUPUS VULGARIS.

This occurs in both sexes, and is more common in young people; indeed, it more frequently begins before the age of thirty, and its progress is likely to be less rapid after that age. It is more common among the poor than among the wealthy. The origin of lupus is still very obscure. Histologically, it has close relations with tubercle, but it does not seem clinically to be associated with tubercular diseases, and it is not hereditary. The subjects of lupus rarely suffer from the typical tubercular lesions, and some tubercular lesions of the skin are recognised which are certainly not lupus. Nevertheless, there is an increasing tendency to regard the tubercle-bacillus as the cause of lupus.

It begins usually as a small pink or brownish-red spot on some part of the face—*e.g.*, the cheek—which gradually enlarges; then small nodules are felt in it, and it becomes slightly raised above the surface. It is rather sharply outlined, smooth on the surface, with a somewhat translucent look, and as it enlarges it may have a more yellow or orange colour. The nodules consist of a vascular granulation tissue—that is, closely aggregated small cells, with numerous capillaries; and the intervening tissue of the cutis and the papillæ are infiltrated with leucocytes, and strings of cells form in the course of the lymphatics. Giant-cells and bacilli identical with those of tubercle are also found.

There is neither pain nor itching. The growth of the disease is slow; it advances irregularly at its edge, and the surface may be more or less scaly; but it ultimately undergoes one of two changes. Either it cicatrises directly, by some of the cells forming fibrous tissue, and others, together with the normal tissues of the skin, becoming absorbed; or the disease proceeds to the surface, the epidermis is involved, the cells degenerate and break down, and an ulcer is formed, which is covered with purulent crusts and scabs. In such an ulcer the edges are raised, the lupus nodules can be recognised in the base, and the pus is thin and scanty. In course of time a lupus patch presents a somewhat irregular arrangement of fresh lupus tissue, of crusted ulcers, and thin, white, ill-defined cicatrices. The patch is generally single and unsymmetrical. It begins most commonly on the face, especially on the ala of the nose, the edge of the lip, the cheek, or the eyelid; or it attacks the ear or the neck. It is much less common on the trunk or limbs, and rare on the scalp. The mucous membranes of the nose, mouth, lips, gums, hard and soft palate, epiglottis, and larynx, are sometimes affected.

Though lupus destroys the skin in which it grows, it is only indirectly destructive of other parts—that is, by pressure and atrophy. Thus, if the disease spreads over the face and nose, in course of time the resulting scars will contract, the lower eyelids are everted, the gums are exposed, the tightly stretched skin compresses and atrophies the cartilages of the nose, and much deformity results. These effects were at one time attributed to actual invasion of the cartilage by the lupus, and its subsequent destruction by ulceration, and so a division into *Lupus exedens* and *Lupus non-exedens* was made. But it is probable that lupus, if it sometimes grows into the subcutaneous tissues, rarely involves fasciæ and cartilages, and never bones, muscles, other deeper structures, or the internal organs. Some cases of *L. exedens* were no doubt tertiary syphilis or rodent ulcer.

In spite of the persistence of the disease, the patients are mostly in very good general health.

Diagnosis.—It is distinguished from all ordinary cutaneous affections by the red infiltration of the skin, accompanied by cicatrisation. It is most likely to be confounded with cancer, rodent ulcer, or tertiary syphilitic ulceration. The diagnosis is most important as between syphilis and lupus; in the former, the edges of the ulcers are not tuberculated, and the skin around is often deeply pigmented, the lesions are not so extensive, but ulcerate more deeply, and often a gumma or deep suppuration precedes the breach of surface; the lesions may be multiple, or accompanied by disease of other parts of the body, such as periosteal nodes, lardaceous viscera, &c.

Treatment.—Local treatment is the only kind that can be with certainty efficient. Cod-liver oil and tonics can at most render assistance in some cases. Lupus is practically a new growth, and must be destroyed. This can be done most effectively by the *cautery*, by *caustics*, and by *scraping* with a metal edge (sharp spoon); less certainly, but still sufficiently in some cases by *scarification*. The principle of the scraping treatment is that the lupus tissue is less resistant than the healthy tissue beyond it. The patient is placed under chloroform, and with the instrument all lupus growth is forcibly removed, while the firm healthy skin is left. With lunar caustic a similar result may be effected; it will break down and destroy more readily the infiltrated skin than the healthy, but it is more likely to want repeating than a well-conducted scraping. Crusts and scabs must of course be first removed, and the new wound may be dressed with zinc ointment. Caustic potash, arsenical paste, and acid nitrate of mercury have been also used, but are very painful, and require careful management to prevent injuring the sound skin. Scarification is performed by making hundreds of punctures into the affected skin, close together and about two lines in depth; and this operation is often repeated. By this means vessels are divided and obliterated, and the new growth is more or less starved. It is obviously more suited to parts that are not ulcerated. In slight cases astringent ointments or the ung. hydrarg. subchlor. may render some service.

The injection of Koch's tuberculin (*see* p. 469) occasionally gave good results in lupus. His " new tuberculin," lately introduced, has also been beneficial, especially in more recent or less indurated lesions.

The great difficulty with all methods is to prevent the recurrence of the disease, which is probably due to the persistence, in spite of apparently thorough treatment, of a small portion of diseased tissue.

LUPUS ERYTHEMATOSUS.

This form of disease occurs mostly in adults, especially between the ages of twenty and forty, and is more common in women than in men. It consists, like L. vulgaris, of hyperæmia, and infiltration with small cells, with the same persistent, slow growth, and partial cure by absorption and cicatrisation, but the sebaceous glands are largely involved, so that they become distended, form comedones, and dried crusts of sebum accumulate on the surface; moreover, the skin does not ulcerate, and the disease has a different distribution from that of Lupus vulgaris, and may be far more extensive. Hitherto the bacillus tuberculosis has not been found in it. It often begins as a red patch on the centre of the nose, and spreads thence right and left over each side of the nose to the cheek, on which it forms a broad patch. It has then much the outline of a butterfly or bat—a very characteristic feature of the disease. The patch is red, injected, desquamating, with obviously enlarged sebaceous glands; the edge is rather sharply defined, irregular, and slightly raised. As it spreads at the periphery the centre becomes pale, and ultimately scars, without preceding ulceration. The ears are sometimes attacked, and the scalp, in which case the hair falls out, and permanent baldness may result. The backs of the hands, and less often the arms, legs, and trunk, are also seats of the disease.

Lupus erythematosus makes but slow progress, and lasts for years; it is free from pain, and itching is slight or none. Some cases have been seen of an acute kind, in which a large part of the body was rapidly covered, and death ensued; but, as a rule, the disease has little or no effect upon the health.

Treatment.—The local treatment should be by mild caustics and stimulants:—Tincture of iodine painted on, ointments of pyrogallic acid (1 in 8), of iodoform (1 in 16), of white precipitate, of yellow oxide of mercury, of zinc, lead, and mercury (*see* p. 926), of litharge (Hebra's diachylon ointment), or of liquor carbonis detergens (1 in 16, Hutchinson). Some cases require milder preparations, others can bear the stronger; in any case the applications must be made regularly and perseveringly over long periods.

TUBERCULOSIS.

The invasion of the skin by tubercle produces other results besides lupus. *Verruca necrogenica* has been already mentioned (*see* p. 453), and a *tubercular ulceration* quite different from that of lupus also occurs. The ulcers may be scattered over different parts of the body; they are round or oval, with pale œdematous granulations, and somewhat undermined edges. Mildly stimulating applications externally, cod liver oil and quinine internally, and the best hygienic conditions, are required.

RHINOSCLEROMA.

This is a dense infiltration of the septum and alæ of the nose, rendering it as hard as ivory, thick, and rigid. The surface is smooth or irregular, the colour normal or brownish-red; the mucous membrane is affected as well, and the orifices may be blocked by its swelling. Not infrequently the adjacent upper lip, and sometimes the cheeks are involved. The change is a small-celled dense infiltration of the corium and papillæ, which is regarded by some as inflammatory, by others as a form of sarcoma.

FRAMBŒSIA.

(*Yaws.*)

This is a specific contagious disease which is prevalent in tropical countries, which runs a chronic course, and tends to spontaneous cure. The eruption consists of one or more papules which are at first about the size of a pin's head, and increase until they measure from a quarter to two inches in diameter. The epidermis gets thin, gives way, and leaves a raw surface, from which a sero-purulent fluid oozes, and this dries into a crust. In shape the tubercles are round, oval, or irregular from coalescence; soft, not sensitive to touch, but itching. They affect the face, lips, nostrils, neck, arms, axillæ, legs, thighs, buttocks, and vulva. If they improve, they contract, dry up, and leave a discoloration of the skin; but they may form large irregular sores, and lead to deep ulceration, gangrene, or stiff joints. The disease affects the sexes equally, but young people more than others, and the negro races more than the white. It is contagious by inoculation of the abraded skin; but a micro-organism has not with certainty been identified.

In the **Treatment** cleanliness is essential. Liq. arsenici et hydrarg. iod. has been found useful internally, and locally astringent and antiseptic applications.

MYCOSIS FUNGOIDES.

In this rare disease, the skin is affected by a number of tumours, the appearance of which is often preceded by erythematous, eczematous, or urticarious patches. The tumours vary from the size of a bean to that of an orange, or larger. They are round, oval, or lobulated, the skin is stretched over them, tense and shining; the skin around them is often infiltrated. After a long duration some of them may shrink and disappear, others ulcerate on the surface and form fungating masses, discharging a clear watery serum. In this stage they are generally painless and free from itching or smarting. The disease lasts months or years, but ultimately the health fails, and the results have been, with one or two exceptions, fatal. Nothing is known of the causes, and little of the nature, of this disease. Arsenic appears to have done some good in the early stages.

MOLLUSCUM CONTAGIOSUM.

This name is given to small tumours on the skin, which are from one-tenth to a third or half an inch in diameter, lenticular or hemispherical in shape, occasionally globular or pedunculated, somewhat irregular or nodulated on the surface, and of a yellowish-white colour. In the smaller tumours there is often a minute opening in the centre—in the larger there are several;

and if the tumour be firmly squeezed, a little milky juice exudes from these apertures. Examined under the microscope, the juice is seen to consist of minute oval glistening bodies, the *molluscum corpuscles* or *bodies*. A vertical section through the tumour shows it to have a structure somewhat like that of a racemose gland. There are lobules separated by fibrous tissue: each lobule has, externally, a columnar epithelium, within this are more oval epidermic cells, and in the centre of each lobule is a collection of the glistening opaque molluscum bodies. The lobules in the larger tumours do not converge to a central duct or opening, but rather lie side by side, and open separately upon the surface.

There is much doubt as to the nature of these growths. That they arise by distension of the sebaceous follicles with cells seems less probable than that they are definite tumours starting in the hair-follicles, or by a downgrowth of the cells of the rete Malpighi. It seems to be possible that they may owe their origin to infection by the minute organisms known as *psorosperms*.

They occur in children and adults; and are situate on the face, arms, or hands, the mammæ of women, and the genitals of men. They have been regarded as contagious, from the fact of their appearing in members of the same family, in mother and baby at the breast, and so on; but it is very difficult to produce them by direct inoculation. They may remain unchanged for long periods, they may suppurate, or they may subside and disappear.

Treatment.—This consists in pressing out the contents, either directly or after incision, and applying a little caustic; or by removal with knife or scissors.

FIBROMA MOLLUSCUM.

This is a soft, flaccid, wrinkled, often pendulous tumour, consisting of a covering of scarcely altered cutis and epidermis, containing a fibrous meshwork with a variable proportion of round cells and of albuminous fluid. The tumours may be very few, or exceedingly numerous; they vary in size from a pin's head to the head of a man, and they occur especially on the trunk. They may apparently be congenital, but are generally first seen in early childhood. According to von Recklinghausen, they are really neuro-fibromata, starting from the fibrous sheaths of the smaller cutaneous nerves, and thence invading the fibrous structures of the vessels, the sweat-glands, and the hair-follicles. In an allied condition there are no separate tumours, but the skin is thickened and overgrown, or lies in large, loose, overlapping folds (*dermatolysis*). Cases of *multiple fibromata* are rare. Removal by the knife is the only possible treatment, and that is scarcely applicable if the tumours are very numerous.

CHELOID.

Cheloid is a growth of the skin and subcutaneous tissue, consisting chiefly of dense bands of fibrous tissue, containing numerous spindle-cells in its earlier stages. Its more common seats are on the chest, over the sternum, in the mamma, on the neck, back, lobules of the ears, and on the limbs. It is usually single. It begins as a flat, smooth, pink nodule, which extends laterally to a considerable size, and becomes paler in the centre, while the skin around is more or less reddened. After a time, bands and ridges, separated by furrows, develop, ranging in various directions across the tumour and into the surrounding skin. By the slow contraction of these bands, much deformity may be caused, and movements of adjacent joints may be seriously restricted. The growth of the tumour is often accompanied by considerable pain and tenderness. Similar growths not infrequently develop in former scars. These have been called false cheloid, but it does not seem that they are essentially different. Cheloid grows slowly, and rarely disappears spontaneously. If removed by a knife or caustic it almost inevitably returns; but it always remains a strictly local disease, invading neither lymph-glands nor viscera. Its severe pain may demand local or general anodynes.

MYOMA. NEUROMA. LYMPHANGEIOMA.

These occasionally occur as cutaneous affections. Neuroma forms multiple, painful small growths in the course of the nerve-fibres of both trunk and limbs (*see* p. 182).

Lymphangeioma is a rare growth, due to dilatation of lymphatic vessels into visible cysts, and overgrowths of the intervening connective tissue. It has been seen by Pye-Smith and others in association with ordinary vascular nævi.

XANTHOMA.

(*Xanthelasma. Vitiligoidea.*)

This affection occurs in two forms. In the more common variety (*X. planum*), one finds generally on each upper eyelid, near the inner canthus, a small sharply-defined patch of whitish-yellow, soft, smooth skin, level with, or scarcely raised above, the general surface. Such patches may remain stationary for years, or may slowly increase, spreading outwards along each upper lid; or other patches appear on the lower lids, and the orbit is completely surrounded by a broad patch of the altered skin. In many cases no other part of the body is affected; but in others spots and streaks of a similar kind appear on the trunk, on the backs and palms of the hands, and the soles of the feet, or on the scrotum. They have also been seen in the mucous membranes of the gums, palate, side of the tongue, larynx and trachea, and in the mucous lining of the bile-ducts (Fagge).

The second form (*X. tuberosum*) consists of firm, rounded nodules, from the size of a pea to that of a nut, occurring on the skin over the elbow, on the knuckles, and on the lobules of the ears. These growths occur, as a rule, in persons of middle age or older, and are more frequent in women than in men.

Ætiology.—The most extensive lesions have been seen in cases of long-standing jaundice, but the connection between the two complaints is not as yet clear. Small patches affecting the eyelids alone are often seen without jaundice, and are thought by Hutchinson to be related to attacks of sick headache. Some curious patches have also been seen in patients suffering from diabetes (*Xanthoma diabeticorum*), but their nature still remains doubtful.

Anatomy.—The flat patches consist of newly-formed connective tissue, in which fat is abundantly deposited. The nodules have a similar structure, but the fibrous tissue is present in greater proportion.

Treatment.—Small patches may be excised, but the removal of larger patches from the eyelids would risk serious deformities. If their removal is called for, caustics are to be preferred.

DISEASES OF THE SWEAT GLANDS.

Anidrosis, or deficiency of perspiration, occurs in fevers, in diabetes, and in some diseases of the skin—*e.g.*, ichthyosis and pityriasis rubra.

Hyperidrosis, or excess of perspiration, may be general or local. General sweating occurs as a result of dilatation of cutaneous blood-vessels, as after exercise, or from emotional causes. Sometimes, on the other hand, it occurs with contracted vessels, as in conditions of collapse or fear. General perspiration has been mentioned in connection with ague, phthisis, pyæmia, and the crisis of acute illnesses. Local excess of sweating occurs from emotional causes and in rickets. A very troublesome form of excessive sweating occurs about the hands and feet, axillæ, and genitals in some persons without any adequate cause. Some sufferers are in deficient general health, but others are perfectly well.

The local application of belladonna liniment or the use of atropine or belladonna internally should be tried. A drop dose of liquor atropinæ will sometimes stop the sweating of phthisis for two or three successive nights. Local sweating may be also treated in the same way, or by some of the methods used for the next complaint.

Bromidrosis.—This is often associated with hyperidrosis—that is, the sweat is both offensive and excessive. It affects chiefly the feet and axillæ; and the odour is probably due to the decomposition of the fatty sebaceous material which is secreted with the sweat. It is not uncommon in young men or young women of the domestic class; and it may be quite independent of the general health. Thin has described a bacterium in connection with it—*bacterium fœtidum*. It is essential to wash the feet thoroughly and frequently, and use astringents and antiseptics. The socks may be dusted inside with finely-powdered boric acid, and should be frequently changed; or with oxide of zinc; or the feet and toes may be carefully strapped with the unguentum diachyli of Hebra, or with ordinary plaster; or smeared with a salicylic ointment, of 2 per cent. strength.

Chromidrosis, or coloured sweat, is a rare affection, and is perhaps sometimes due to indican. But the possibility of its being feigned should always be remembered.

Hæmatidrosis, or sweating of blood, also quite rarely occurs, mostly in highly neurotic people.

Uridrosis is the name given to some cases in which the sweat has crystallised on the surface, and the crystals have been found to contain urea and salts. It has been seen in Bright's disease (*see* p. 796) and in the stage of collapse of cholera.

Dysidrosis has been already mentioned (*see* p. 920).

Sudamina.—These are small transparent vesicles, not larger than a pin's head, occurring as a result of profuse sweating, and due to the elevation of the most superficial layer of the epidermis by accumulated sweat. There is no doubt a relative obstruction of the sweat-ducts from the rapidity of the secretion. They are found most abundantly on the chest and abdomen, but from their perfect transparency may be better felt than seen. The vesicles dry up, and leave a few branny scales, the remains of the detached epidermis. They are most common in phthisis and in enteric fever.

Miliaria.—These are vesicles, produced by some similar mechanism, but accompanied by inflammation. They are surrounded by a red areola, and contain a yellow turbid alkaline fluid or actual pus. It is not uncommon to see these in the course of rheumatic fever.

According to Crocker, the *strophulus*, or *red gum*, of children is nothing more than miliaria, consequent upon sweating from too much wrapping up. The same author classifies under the term *Miliaria papulosa* the disease known as prickly heat, which has been commonly regarded as a papular eruption, lichen tropicus. It affects chiefly the trunk and thighs, is accompanied by severe itching, and does not materially influence the general health.

DISEASES OF THE SEBACEOUS GLANDS.

SEBORRHŒA.

Excessive secretion of sebum occurs in two forms—*Seborrhœa oleosa* and *Seborrhœa sicca*. In the former the face appears constantly greasy, or moist, and consequently shiny; if the finger touches it, it is obviously moistened by the greasy secretion; the face, moreover, gets readily dirty from the adhesion of the particles of dust floating in the air. It is most frequent about the forehead and nose, but occurs on all parts of the face. It varies from time to time, being, perhaps, aggravated by gastric troubles, and by general ill-health.

In *Seborrhœa sicca* the secretion is less fluid, but consists rather of solid fatty substances mixed up with epithelial particles. It is not uncommon on the heads of infants, forming thick brownish or yellow-green plates or patches rather firmly adherent to the scalp. In older persons the secretion forms minute whitish branny particles, which develop over the whole head, are readily detached, lying loose among the hair, or falling on to the shoulders, and are known as scurf or dandriff. Formerly this was described as *pityriasis capitis*. If it continues for long it may lead to a weak growth of hair, or actual falling out. Either form may be accompanied by a slight redness of the scalp at the affected part. Micro-organisms have been found in seborrhœa, and there are reasons for believing that it is contagious.

Treatment.—The general health should be attended to. In seborrhœa sicca the crusts should be removed, and astringent and mild stimulant ointments may be used, such as those of zinc oxide and oleate, mercurial ointments, or carbolic oil.

Seborrhœa corporis.—This eruption (formerly called *lichen circumscriptus*) consists of small flat dusky red papules, combining to produce circular patches a quarter or three-eighths of an inch in diameter, which may clear in the centre, and ultimately form rings of a larger size. By running together they result in scalloped and gyrate figures. The papules are often covered with a yellowish-brown scale. They occur almost exclusively over the sternum and on the back between the shoulders, but may extend from the middle line in front over the pectoral regions. The eruption itches slightly, but otherwise causes little discomfort, and may be only noticed when the patient consults his doctor for some other complaint. The skin is often greasy, and the patients are found to wear flannel shirts or vests. It is more frequent in men than in women.

The situation, the annular shape, and the frequent yellowish tinge are characteristic. Only if very extensively developed in a gyrate form could it be suspected of having a *syphilitic* origin.

Treatment.—Tar or creosote ointment, and glycerine of borax or thymol, cure it speedily; but it readily returns if the local conditions are not altered by frequent washing, and suitable changes of underclothing.

Seborrhœa congestiva is a name given by Hebra to *Lupus erythematosus.*

COMEDO. ACNE.

Not uncommonly the sebaceous follicles get blocked by their secretion, and thus lead to a prominent papule on the skin (*comedo*), or to inflammation around the distended follicle (*acne*).

The comedo is commonly seen as a whitish conical swelling on the forehead, cheeks, or nose, with a minute black spot on the summit. The swelling is due to the accumulated sebum; the black spot is adherent dirt. It is frequently accompanied by seborrhœa. If pressure be made upon the base of the papule with a finger-nail on either side, a plug of sebum is extruded, and occasionally in this sebum can be found an acarus, the *Demodex folliculorum;* but there are always bacilli and cocci in great variety.

Comedones may persist sometimes without much change, fresh follicles being involved from time to time; but generally some of them become inflamed so as to constitute acne, and often the great majority of the lesions are quite early of this nature.

In *acne* the papule tends to be larger, is conical in shape, pink or red in colour, and as suppuration takes place within the follicle, a pustule forms at the summit. Eventually the pustule bursts, and the redness subsides, leaving no trace; but the lesion is sometimes so extensive, and the suppuration so deep, that a well-marked scar is left. This is especially likely to be the case where the papule is irritated by the friction of the clothes, as for instance, on the back and shoulders.

The ordinary form is called *Acne vulgaris;* large hard inflamed papules constitute *A. indurata;* those which have suppurated freely, *A. pustulosa.* Comedo was formerly called *A. punctata.*

Acne affects especially the face, chest, and shoulders, the back of the chest and shoulders often presenting the largest pustules and the most numerous and extensive scars, while the front of the chest is the least involved.

It is a disease of early adult age, or puberty; beginning in young men before the beard has begun to grow, and commonly subsiding after it has grown, and rarely lasting after the age of thirty. It occurs also in young women, but less commonly and less severely than in men. Beyond these relations of age there is not much positively known as to the cause of acne. Exposure to dirt and grease, as amongst some classes of artisans, especially those having to do with tar, no doubt produces it by direct obstruction of the sebaceous follicles; but where that cannot be alleged as the cause, its origin is mostly quite inexplicable. A certain amount of anæmia is not infrequently present.

Treatment.—Comedo should be treated by thorough washing with soap and water night and morning, and drying with a rough towel, by which means already some of the plugs will be removed. Others may be pressed out by placing over the top of each papule a watch-key, or other key, and pressing firmly until the plug is extruded. Some special instruments have been devised for this purpose. Both in this and in acne any condition of ill-health may be dealt with, and it is common to give mild laxatives and iron, as they are believed to be of value even when no anæmia or digestive fault is in question. The sulphates of iron and magnesia in peppermint-water form a very good mixture for this purpose. Other tonics may be suitable in special cases, and the diet may as well be regulated. Where there is definite acne, the washing and friction may still be carried out; pustules should be opened with a needle or lancet-point, and some stimulant preparation should be applied. This may be left on only during the night, as the course of the disease is always slow, and the inconvenience of having ointments on the face during the day may be respected. Sulphur is one of the most useful of local remedies: it may be used as iodide of sulphur ointment, which is effectual, but temporarily stains the skin; sulphur suspended in liquor calcis; ointment of precipitated sulphur (2 or 3 drachms in an ounce of vaseline); or a lotion of sulphide of potassium (1 to 40). If there are many pustules, mercurial ointments are also valuable, such as white precipitate, or the acid nitrate: but with much dermatitis between the acne pustules, soothing remedies, like zinc and lead lotions, may have to be temporarily employed. In any case, the treatment must be perseveringly continued.

ACNE ROSACEA.

(*Gutta Rosea.*)

This differs in many respects from ordinary acne, though inflammation of the sebaceous follicles forms part of it. Pye-Smith regards it as an erythema, or the result of recurring erythema. It occurs in men more frequently than in women, and seldom before middle life. The most common cause is the frequent use of alcohol, or the kind of indigestion which is the result of excessive drinking and over-feeding; in women, ovarian and uterine disturbances are frequently the cause. It begins as an erythematous redness affecting the nose; repeated attacks lead to infiltration and permanent thickening of the skin. It spreads to the cheeks, the centre of the forehead, and the chin. The minute vessels become dilated, and are visible on the surface; the sebaceous follicles are distended with secretion, and inflame, so that eventually the affected part shows a deep red swollen and thickened skin, with dilated venules, and papules, pustules, and nodules of various sizes. The nose is especially the subject of great hypertrophy of the tissues, and may form a large lobulated tumour of an inch or more in diameter (*rhinophyma*). Congestion and inflammation of the deeper layers of the corium, accumulation of sebum, with inflammation of the sebaceous glands, and abundant growth of connective tissue, are the leading histological changes.

Diagnosis.—It is distinguished from *Lupus erythematosus* by the absence of scabs and the presence of pustules; and from *syphilitic* eruptions by the symmetry, and the absence of other characteristic lesions.

Treatment.—The errors of digestion must be corrected. Alcohol should be forbidden, the diet carefully revised, bismuth, alkalies, or bitter tonics administered, and a regular action of the bowels ensured. The local treatment may be similar to that of acne vulgaris, but the stimulants must be milder. Dilated vessels may be incised, and touched with silver nitrate, but large excrescences will require removal with the knife.

The name *Acne varioloformis* is given to a rare pustular eruption which occupies the forehead, scalp, and temples, and leaves scars deeply pitted like those of small-pox. There is still some doubt as to its true position in classification.

MILIUM.

This is a small bright white round tumour the size of a pin's head or slightly larger, which results from complete obstruction of the duct of a sebaceous gland. Such little tumours occur on the forehead, eyelids, cheeks, and genitals. Occurring in children they were formerly called *Strophulus albidus*. Sometimes they occur in great numbers on thin cicatrices, especially those of lupus. The contents are cholesterin and fatty material. They can be treated only by puncturing the skin and squeezing out the contents.

SEBACEOUS CYSTS.

(*Wens.*)

These are retention cysts arising from obstruction to the ducts of the sebaceous follicles. They are most common on the scalp, but occur on the eyebrows, face, or neck, and more rarely on the trunk or limbs. In size they vary from that of a pea to that of a nut or an orange. They are hemispherical, or more globular, uniform and smooth. The skin looks thin, and often presents well-marked vessels ramifying over it. The contents are semi-fluid, or pasty, and consist of animal fats, albumen, epidermic cells, cholesterin, and earthy salts, enclosed in a capsule made up of layers of epithelial cells and fibrous tissue. The **Treatment** consists in incising the cyst, squeezing out the contents, and tearing out, or dissecting out, the cyst wall entirely.

DISEASES OF THE HAIR AND HAIR FOLLICLES.

The hair may be developed to an excessive degree as a congenital phenomenon; this is very rare and is called *hirsuties*. Deficiency or falling of the hair, known as *alopecia*, is, on the other hand, exceedingly common. The following three conditions are quite rare :—*Trichorrexis nodosa*, in which some of the hairs are found to present little nodules or thickenings due to the splitting up of the cortical fibres; *monilethrix*, in which the hair looks beaded, and breaks readily at the internodes, so that it is only two or three inches long all over the head; and *lepothrix*, in which the hairs are brittle, and present irregular masses on and around them.

The most common *change of colour* in the hair is that known as *canities*—the hair gets successively grey and white. This is a senile change that may occur prematurely. But occasionally sudden whitening of the hair occurs after fright, intense emotion, or in consequence of neuralgia. It is probably due to the development of air-bubbles, which conceal the pigment. The colour sometimes returns spontaneously, but nothing can be done for it.

Of the above ailments, *alopecia* will be more fully described, and afterwards *sycosis*, or inflammation of the hair-follicles. The hair is also involved in some of the parasitic diseases of the skin.

ALOPECIA.

Baldness is most familiar to us as it appears in old age (*senile alopecia*), though it occurs also in quite young people (*premature alopecia*). Different degrees of atrophy of the hair-follicles and structures of the skin have been found in old cases of baldness, but its immediate antecedent is doubtful. Nor is much known of the cause of premature baldness, though it has been variously ascribed to mental exertion, wearing tight hats, insufficient lubrication of the scalp, &c. It is certainly sometimes hereditary, and it may be hastened by affections of the skin of the head, such as seborrhœa sicca, or eczema. The hair thins first at the back of the vortex, and at the front parts of the temples. Ultimately there is only a fringe left round the temples and occiput. A temporary alopecia is caused by transient interference with the nutrition of the hair-bulbs; thus the hair falls not infrequently after fevers, after erysipelas of the scalp, in the second stage of syphilis, and in the parasitic diseases which will presently be described.

Premature loss of hair is practically incurable. Restoration of the hair after fevers, &c., may be hastened by the use of tonics internally, faradising by means of the wire brush, and by the use of stimulants, such as oil of mace, cantharides, and ammonia, which will be again mentioned under the next complaint.

ALOPECIA AREATA.

(*Area Celsi.*)

This peculiar form of baldness occurs at almost any age, but is commoner in children and young people. The sexes are about equally affected, and in the majority of cases there is no indication of ill-health. Quite unexpectedly it may be noticed that there is a bald spot on the head, the size of a sixpence or larger, and this gradually extends. The spot is circular, and, as nearly as possible, perfectly bald, smooth, and shining. But one feature is very constant—namely, that just at the edge of the patch, and less commonly in its middle, are observed a few—from one to ten, or more—short, dark stumps, from $\frac{1}{8}$ to $\frac{1}{4}$ inch long, and thicker at the free end than at the root. If extracted and placed under a microscope, the free end is seen to be brush-like and broken up into its constituent parts; while there is no bulb, but only an atrophied root. No indications of a specific fungus can be found. Other bald patches may form on the scalp adjacent to the first, or elsewhere, and two or more may coalesce to form an irregular figure. The patches persist some months or a year, and gradually become covered with hair, so that complete recovery takes place.

The **Pathology** is still obscure. Histological examination of the skin has shown atrophy of the hair-follicles, and round-cell infiltration of the outer root-sheath, the surrounding skin and subcutaneous tissue. The views held with regard to it are—(1) that it is a parasitic disorder, chiefly on the evidence of alleged cases of contagion, and of its occasional association with ordinary ringworm; (2) that it is a disorder of nervous influence—atropho-neurosis; (3) that it is the result of changes in the sebaceous glands.

The **Diagnosis** presents no difficulties, as ringworm is rarely so completely bald, and the stumps of hairs have a different appearance.

Treatment.—This consists in such tonic treatment of the general health as may seem indicated, and the application of local stimulants and irritants—such as ammonia liniment; or tr. cantharidis in water (1 to 8); or acetum cantharidis or tr. cantharidis ʒss., carbonate of ammonium gr. xl., and sp. rosmarini ʒss., with water to 8 ounces; or linim. myristicæ of Guy's Hosp. Pharmacopœia (expressed oil of nutmeg, 1 part, olive oil, 3 parts). One of these should be rubbed in every night, to the extent of producing slight redness only. The faradic brush and occasional blisters may also be employed.

UNIVERSAL ALOPECIA AND CONGENITAL ALOPECIA.

In the former the hair falls from every part of the body, scalp, eyebrows, axillæ, and pubes, so that not a hair is left. As a rule, no cause can be assigned, and the cases are incurable.

In the latter, the nails are wanting as well as the hair. These cases may be hereditary.

SYCOSIS.

This is a chronic inflammation of the skin of the beard and ha parts of the face, beginning in or around the hair-follicles, a occurring exclusively in male adults. The infiltration is de seated, pus forms in the follicle, and the hair is loosened. If pus escapes it dries up into an adherent crust. The pustules m be at first isolated, but the intermediate skin is involved, and c siderable infiltration takes place, the part being red, irregular, a nodulated, with pustules and adherent crusts. It appears to contagious, no doubt by means of the pus-organisms.

A somewhat similar but rarer condition arises from action of the ringworm fungus (*see* p. 965). In this the extr tion of the hairs is less painful, and the fungus can be reac recognised under the microscope. Sycosis may be also confoun with eczema, but in this there is more itching, the lesions are m superficial, and generally extend beyond the limits of the ha parts.

Treatment.—The disease is most obstinate, but may be cured perseverance. The hair must be cut close, the crusts must detached by softening with poultices or oiled lint, and the h must be removed.

Epilation may be begun with the hairs that are loosened inflammation, and only subsequently, if necessary, it may extended to others. The inflammation may be allayed by the of lead and zinc ointments, applied thickly on linen. As the l grows it must be constantly cut, and epilation steadily perseve with. When the infiltration has subsided, and the pustules less numerous, the hair may be shaved. Months of steady tre ment may be required.

VEGETABLE PARASITES.

The following diseases of the skin are due to vegetable paras belonging to the class of *Hyphomycetæ*—*Tinea versicolor*, *E thrasma*, *Ringworm*, and *Favus*. The parasites, or fungi, consis jointed rods or threads, the mycelium, and round or oval bod the conidia, or spores. Their average size is greater than t of the bacteria, and they can generally be well seen wit moderately high power of the microscope (one-fifth in after the hair or epidermic scales with which they connected has been rendered translucent by liquor pota These fungi have also been cultivated on various media bacteriological methods, and their distinctive characters more fully brought out.

TINEA VERSICOLOR.

This is a common affection of the skin, no doubt produced by contagion, and fostered by warmth and moisture. It is more frequent in men, and especially in those who wear flannel underclothing. It is not often conveyed directly by contact—for instance, from husband to wife.

The disease begins as a small circular spot, of a yellowish-brown colour, which is slightly raised above the skin, and from which a few whitish scales can easily be detached by scraping with the finger-nail or a scalpel. The patches extend, and fresh ones form, so that soon a large part of the chest, where it is commonly seen first, is covered with a brown, or brownish-yellow, irregular patch, with a convex or scalloped margin; and on the healthy skin adjacent are numerous small isolated patches from a quarter to half an inch in diameter. The disease occurs only on covered parts, and is most abundant on the front and back of the chest and the abdomen. The scrapings examined in potash under the microscope show epithelial plates with the characteristic fungus—*Microsporon furfur*—forming a network of branching mycelium threads, with little groups of the relatively large conidia, like bunches of grapes. The affection does not cause much trouble beyond some itching, and is frequently ignored by the patient; but the great extent which the discoloration may sometimes reach has led to its being mistaken for Addison's disease and other pigment affections. The peculiar colour, the convex edge, and the ready desquamation of the surface should be quite distinctive, and the diagnosis is at once confirmed by the microscope.

Treatment.—It is quickly cured by rubbing in lotions of sodium hyposulphite (1 in 8) or of sulphurous acid, or finely powdered borax. It may, however, return if the same underclothing is used without thorough washing and disinfection.

Erythrasma is an allied disease of the epidermis described by German authors. It produces rough, brown scales, occurs chiefly between the scrotum and thighs, in the axillæ, and mammary folds. It is due to a fungus, the *Microsporon minutissimum.*

RINGWORM.

The diseases which are commonly included under this term are *Tinea tonsurans*, or ringworm of the head; *Tinea circinata*, or ringworm of the body; *Tinea marginata*, or Burmese ringworm;

Tinea sycosis, or ringworm of the beard. Until recently, th were believed to be all dependent upon one organism, the Tric phyton tonsurans. It is now known, largely through the work Sabouraud, that there are two, if not three, different fungi wh are concerned in the production of tinea. It will be better describe the clinical features of the various forms first, and st what is known of the organisms afterwards.

Tinea Tonsurans, or Ringworm of the Head.—This disease is great scourge of schools, and allied institutions, among the poo classes. It is frequent in children, rare in infancy, and not eas caught by adults. It spreads by contact, and by the use of ha caps, brushes, combs, and towels in common.

It generally first appears as a round patch, on which t growth of hair is much thinner than elsewhere. On cl examination the skin is seen to be pink, perhaps a little swoll and covered with minute branny scales. Besides the thi scattered long and healthy hairs are seen a number of brok stumps of hair, opaque, black or dark brown in colour, twist and bent. If an attempt be made to extract one of these brok hairs with a pair of forceps, it will almost certainly break short; if then placed under the microscope, and moistened wi a drop of liquor potassæ, it will be seen that the natural struct of the hair is unrecognisable. The substance is quite opaque, a the hair seems to be converted into a mass of fungus spo (conidia). This is, however, only a sheath of spores surroundi the hair, which is itself occupied chiefly by mycelium tubes runni in a longitudinal direction. These may be more readily observ in hairs which are less completely diseased.

The patch spreads by the implication of hairs at its circu ference, and fresh patches form in other parts of the sca As these enlarge they become more completely denuded of lo hair, though they nearly always present a considerable quant of the short stumps which have been described; and these m be surrounded and mixed with scabs, crusts, or sebaceous matt or with a fine whitish powder, of which probably the fung elements form a part. Sometimes the inflammation is mu more decided; the hair-follicles inflame, coalesce, and a red pink swelling occurs, which is soft and boggy to the touch, d charges pus from a few points, and tends to become quite ba from the loosening and falling out of the hairs. This conditi is known as *kerion*. In most cases, however, the inflammati does not reach any considerable degree. The patches spre slowly; some may heal in the centre as they extend at t edges; the patches may coalesce, and nearly the whole sca may be affected. Sometimes, on the other hand, one or tw patches persist, without improvement, but without spreadin The disease may last for years, but eventually dies out, and t hair is perfectly restored, except possibly over patches of very severe kerion.

There is some difference of opinion as to the exact way in which the disease begins. Probably the spores invade the hair close to the scalp, and push down towards the bulb. The hair in the follicle is thus weakened or destroyed, and as it is forced outwards by the newly-formed epithelial plates, it breaks off. The newly-formed epithelium is, in its turn, invaded as soon as it gets into the horny condition. It has been shown by Thin and myself that the fungus only invades the hair itself, and cannot be found in the cells of the root-sheath or in the structures of the hair-follicle.

Tinea Circinata, or Ringworm of the Body.—This occasionally co-exists with tinea tonsurans, but often occurs alone. The fungus invades the epithelial scales and downy hairs of the skin, and produces a circular patch, from half to one inch in diameter, slightly raised above the surface, sharply defined, pink in colour, often papular, and covered with fine scales. If the surface be scraped with a scalpel, and the scrapings be placed, with a little liq. potassæ, on a glass slide, the mycelium and spores of the fungus will be seen. The patches increase by extension at the circumference, and may, as they spread, heal in the centre. Occasionally a few vesicles form on the surface from the irritation of the parasitic growth; thus in part justifying the former name, herpes circinatus. They occur on the face, neck, and arms most frequently; they are, as a rule, few in number, and may be solitary.

Tinea Marginata (Eczema Marginatum, or Burmese Ringworm).—This is confined to adult males, and occurs chiefly about the inner sides of the thighs, genitals, and groins of those who are constantly sitting, such as horsemen and cobblers. It begins in spots or rings, which, spreading at the margin and recovering at the centre, unite together, and ultimately extend on both sides symmetrically. It forms a broad yellowish or brownish-red band, which runs in a curved or gyrate form along the inner side of the thigh and scrotum to the inner side of the buttocks, and over the groin and lower part of the abdomen. The lesions are much more extensive, more inflamed, and more obstinate than those of tinea circinata.

Tinea Sycosis, or Ringworm of the Beard.—The hair-follicles of the chin and cheeks are here inflamed by the presence of the fungus; they suppurate, and the hairs become loosened. Induration and swelling of the intermediate skin also occur. It differs from sycosis, already described (p. 962), in that it first attacks the hairs, and loosens them early, so that their extraction is painless; it spreads more rapidly, and produces deeper infiltration. Microscopic examination shows the fungus, in which the mycelium is more abundant than in the spores.

Tinea of the Nails.—The invasion of the nails by a fungus is called *onychomycosis*, and this may be a ringworm or favus, either of which is probably conveyed by scratching other affected parts. The nail tends to become elongated and curved over the end of the finger, with a thick edge, rough uneven surface, and dirty yellow colour; it is also brittle and readily splits. If fragments or scrapings are soaked with liq. potassæ, and examined under the microscope, chains of spores of the fungus are seen.

The Fungi of Ringworm.—The point upon which a separation of these fungi is based is the size of the spores. Tinea tonsurans is, at least in England, almost entirely due to a variety in which the spores are small, measuring from 2 μ to 4 μ. It is hence called *microsporon*, and appears to be identical with the *Microsporon Audouini*. As usually seen, the shaft of the hair is penetrated by the mycelial threads and the spores form a dense sheath, five or six spores deep, around the hair, and this sheath is continued beyond the follicle on to the stump or fragment of the hair which still remains. The common ringworm, caused by the *microsporon*, is almost confined to children, is very contagious and very obstinate, so that months or years may elapse before it is cured; but it ultimately gets quite well.

The other ringworm fungus is named *Trichophyton megalosporon*, and two varieties of this are described: *T. endothrix* and *T. ectothrix*. The spores measure from 3 μ to 6 μ, commonly 5 μ, and are thus larger than those of microsporon. In Tricophyton endothrix the spores and mycelium are contained entirely within the shaft of the hair; in T. ectothrix the spores form a sheath outside the hair (as they do in microsporon), and only the mycelium tubes lie within it. Cases due to megalosporon are much more amenable to treatment, and those due to the ectothrix variety more than those due to endothrix. As to the further relations between the fungi and the clinical forms of disease very different statements are made, which are no doubt in great part explained by the different prevalence of the fungi in countries remote from one another, the experience of Paris being different from that of London. Observers of the disease in England (Morris, Fox, Adamson) show that the microsporon causes the large majority of cases of ringworm of the head; and that it is present in kerion, which Sabouraud attributed with all other pustular forms to a megalosporon. Trichophyton megalosporon endothrix is the chief cause of Tinea circinata, and T. megalosporon ectothrix is present in a few cases of Tinea tonsurans, in Tinea sycosis, and in tinea of the nails (onychomycosis). These fungi occur also in domestic animals, and though contagion is, especially in ringworm of the head, mostly from child to child, it is believed that the disease may sometimes be caught from the horse, dog, or cat.

Treatment.—The treatment of *Tinea tonsurans* requires to be carried out with the greatest pertinacity, and even then six, twelve, or more months may elapse before the disease is perfectly cured. The difficulty lies in the fact that the spores multiply in the hair-follicle, and are therefore, to a large extent, protected from the parasiticides employed. The first thing is to cut the hair quite close, and remove all crusts and scabs by soaking with oil and subsequent removal and washing. The extent to which hair is removed may vary with circumstances. Complete shaving is best; but where it is desirable to save appearances, a fringe of hair may be left all round the head, or with a single patch the hair may be close cut or shaved for an inch or more round it. Parasiticides must then be regularly and constantly used. The patch should be washed clean with soap and water night and morning, and the ointment or paste well rubbed in.

The following are some of those most commonly employed:—Mercurial applications, such as ung. hydrarg. ammon.; oleate of mercury, from 3 to 10 per cent.; glycerinum acidi carbolici, or the same with a larger amount of carbolic acid; sulphur ointment; creosote; tincture of iodine; thymol or turpentine; or some of these may be combined together, as sulphur 2 and carbolic acid 1 in 16 of lard or vaseline, or iodine 1 and creosote or oil of cade 3 parts. Boric acid ʒj. with methylated ether ℥x. oil of rosemary ʒij. and methylated spirit to ʒxl. is recommended by Aldersmith. Harrison suggests an ointment composed of caustic potash 9 grains, carbolic acid 24 grains, in ½ oz. each of lanolin and ol. cocoæ. Formalin in 40 per cent. solution, rubbed in vigorously with a brush ten minutes on alternate days for four days, is sometimes efficient, but it seems to be painful (Salter). Kerion may be treated with weak lotions of lead or boric acid, and gradually subsides; it should never be incised.

In order to test the effect of treatment the hairs must be examined from time to time, but no case can be considered cured until a good crop of hair has grown over the whole scalp, and even then a very careful search must be made for still active disease, which may be shown by a broken and twisted hair, or a small brown scaly spot.

During the treatment of ringworm great care should be taken to prevent its spread to other children. The patient should use a separate brush and comb, and towels. The head should be covered with a cap, which may be freshly lined with a piece of tissue paper every day, the old piece being destroyed.

Tinea circinata is easily cured by the use of ung. hydrarg. ammon., oleate of mercury, tincture of iodine, sulphurous acid in solution (1 part to two or three of water), a weak carbolic acid glycerine, hyposulphite of sodium (1 in 8), or some other not too strong parasiticide.

Tinea marginata and *Tinea sycosis* require the constant use of similar parasiticides; and epilation must be used for the latter.

In *onychomycosis*, the nails should be scraped thin, softened with alkaline solutions, and soaked in lotions of sulphurous acid, sodium, hyposulphite of sodium, or mercury perchloride (2 grains to 1 ounce of water); or they may be frequently painted with carbolic acid.

FAVUS.

In this disease, rare in England, the fungus attacks the epidermis and the hair-follicles: it may at first form patches like those of ordinary ringworm, but soon there appears a small bright yellow circular disc, with a depressed centre, and a gradually thinning margin. This, the "favus cup," is caused by the fungus elements separating the layers of the epidermis and lifting them up, except at the central point where the hair-follicle joins the skin. This characteristic lesion may occur on the scalp, or on any other part of the body, the forearm for instance, determined by contagion; and it is conveyed not only from man to man, but to man from domestic animals—rabbits, dogs, cats, and others. When numerous cups have formed, they become aggregated together, and form a thick continuous yellow crust, with an irregular honeycombed surface, giving forth an offensive odour resembling that of mice. The hair-sacs are destroyed, the hairs fall out, and baldness results; moreover, the favus masses often become a nidus for pediculi, and eczema and impetigo complicate the original lesion. If the masses are examined under the microscope after soaking in potash, the mycelium and spores (conidia) of the *Achorion Schönleinii* are seen. The conidia are larger and more varied than those of the ringworm fungi, and the mycelium is shorter and more jointed.

The *nails* may be also invaded by the fungus of favus. They present an appearance similar to that in ringworm (*see* p. 966), and sometimes a distinct cup forms under the nail. The nail may be examined in the same way.

Treatment.—The crusts must be softened by oil or poultices, and removed; parasiticides must then be perseveringly employed, and the hairs must be steadily epilated. The nails, if affected, may be treated in the same way as when they are diseased by the trichophyton. The disease is very obstinate, and after apparent cure often breaks out again. It is well to care for the general health by good food and tonic medicines.

ANIMAL PARASITES.

SCABIES.

Scabies, or itch, is a multiform disease of the skin, due to the irritation of the itch-acarus, *Sarcoptes hominis*. The female acarus is oval in shape, $\frac{1}{70}$ inch in length, presents in front four little nipple-shaped processes provided with suckers on stalks, and behind four similar processes provided with long bristles. The male is smaller, has four suckers in front, two suckers and two bristles behind. The female after impregnation bores her way under the skin in an oblique direction, so that as the superficial layers of the epidermis are detached by friction, she still remains the same depth from the surface. As she proceeds she lays her eggs, one or two daily, it is said, and she may thus burrow through the skin in an irregular line for a third or half an inch. Such a burrow (*cuniculus*), or "run," may be recognised on the surface of the skin by the following points:—At one end, the epidermis is broken or frayed, and the free edges are dirty; at the other end is a minute white pointed elevation, in which the acarus lies; the burrow itself, between these points, is a sinuous black line. The whole burrow may be snipped off with a pair of scissors curved on the flat, or shaved off with a scalpel; and if it be then moistened with liq. potassæ and examined, there will be seen the female acarus, and behind her, filling the burrow, her eggs in every stage of incubation, with minute black spots of excremental matter among them. As the skin desquamates, the most developed ova come to the surface, and are hatched. The male does not burrow, but remains on the surface, where he may be sometimes accidentally caught.

As a result of the invasion of the itch-acarus, there are considerable itching and dermatitis of variable extent and character. The itching is mild or severe, but not generally so bad as that caused by pediculi. It is worse at night when the patient is warm in bed. The dermatitis consists of papules, vesicles, pustules, or even bullæ, which generally appear in the neighbourhood of the burrows, but also in parts more remote. Not infrequently a vesicle or pustule forms in the burrow itself. In addition to these lesions, patches of eczema, impetiginous crusts, and urticarious wheals are often present. There is, thus, very great variety in the lesions in different cases. In some, the burrows are numerous, with few inflammatory lesions; in others, vesicles and pustules are abundant, and burrows are with difficulty found. In some instances there are more papules, in others more pustules. The parts of the body especially liable to the attacks of the acarus are the skin between the fingers, the front or inner side of the wrist, the front of the forearm, the ankle and foot, the axilla, the groin, and the genitals, the inner side of the thigh and the nates; and the eruption spreads beyond these parts, on to the abdomen from the groin, or along the inner side of the leg or thigh. The back, shoulders, and chest are but little affected; and the face, neck, ears, and scalp nearly always escape. The occupation of the patient may have influence upon the localisation. Hebra was in the habit of recognising the occupation of a cobbler by the predominance of itch-lesions on the buttock. If the employment involves the immersion of the hands in materials (oily or otherwise) antagonistic to the acari their presence in this typical situation will, of course, be prevented. In children, the lesions are more widespread, the feet and ankles are commonly affected, and pustules are frequently present.

Diagnosis.—The localisation of the lesions is an important guide to diagnosis. An itching eruption of mixed papules, vesicles and pustules, occurring mainly about the fingers and wrists, and also in the other situations mentioned, should lead to a careful search. If a burrow can be found, the minute white elevation at the cleaner end should be looked for, and its epidermis carefully scratched through with the point of a needle; the acarus may then be picked out, as it readily adheres to the surface of the needle. If this cannot be done, it is best to snip off the whole burrow, and to examine for ova or fragments of the acarus under the microscope. Finally, where there has been much inflammation, so that burrows cannot be found, the crusts may be removed, boiled in solution of potash or soda, the fluid allowed to settle in a conical glass, and the sediment examined for fragments of acarus.

Treatment.—Itch is generally quickly cured by the free inunction of sulphur ointment. This should be done at night, and the ointment should be left on, covered by suitable clothing, until the morning, when it may be washed off in a hot bath. The same process may be repeated on two successive nights. The disagreeable odour of sulphur ointment may be lessened by the addition of balsam of Peru. For some skins, sulphur ointment is too irritating, and requires dilution. An ointment of storax (prepared storax 145 grains, methylated spirit, 3 fl. drachms, lard 1 ounce, Guy's Hosp. Pharm.) is also a more pleasant, less irritating, and yet effectual preparation. Vlemingkx' solution may also be used, or sulphur baths (potassium sulphide 4 ounces in 30 gallons of water). It must be remembered that the itching may continue for some time after the acarus has been destroyed. In order to prevent the recurrence of the disease, it is necessary that new clothes should be worn, and that the old ones should be quite disinfected by baking before being worn again.

PHTHEIRIASIS.

Pediculosis.

The pediculi or lice which infest the human race are of three species—the *Pediculus capitis*, or head louse; *P. corporis, vel vestimentorum*, the body louse; and *P. pubis*, or crab-louse.

PEDICULUS CAPITIS.

The head louse is about 2 mm. long by 1 broad, and breeds in the hair of the scalp. Its ova are found adherent to the hairs, and are called nits. They are about ½ mm. in length, whitish, somewhat conical in shape, with the apex always towards the scalp; and they are fixed to the hair by a cylindrical sheath of chitinous material, extending some little distance beyond the apex. The irritation of the pediculus leads to constant scratching and pustular eczema (or contagious impetigo, Crocker). This eruption is most common and severe in the occipital region, and the sub-occipital glands are mostly enlarged as a consequence, and may suppurate.

The **Diagnosis** is not difficult. If the pediculi are not at once seen, the nits, which are readily distinguished on careful examination from scurf, will show at once that there are or have been pediculi. The position of the crusts at the back of the head is also strongly in favour of pediculi.

Treatment.— The insects can generally at once be destroyed by the use of white precipitate ointment; but if there is much eczema or impetigo the hair should be cut over it, and the crusts removed. Hebra recommended pouring petroleum over the head as a rapid and certain means of killing the animals. The nits are not easily detached from the hairs; the cement is very resistant to acids, alkalies, and spirit, but dilute acetic acid (1 in 4) is said to soften it. They can always be slid off the hair, or may be sometimes combed off; but if very numerous, it is probably best to cut the hair.

PEDICULUS VESTIMENTORUM.

This species is larger than the head louse, being from 2 to 3 mm. long, and 1 to 1½ broad. It only occurs on the parts covered with clothes, and chiefly about the back and front of the chest, loins, and abdomen. Occasionally the upper arms, thighs, and even legs may be attacked, but never the face or the hands. The body louse causes a severe prurigo, with intense itching, whic leads to proportionately violent scratching. All the lesio described under Prurigo (*see* p. 931) may occur, papules, bloo crusts, scratch-marks and the elongated scars which result fro them, and after a time, intense pigmentation.

It occurs especially in old people, amongst the poorer classe who have been allowed, from want of proper attention, to laps into conditions of filth and neglect. The disease has then bee called *Prurigo senilis*, and also *vagabonds' disease*.

In its milder form it presents only scattered papules, bloo crusts, and scratch-marks over the upper part of the back an shoulders.

Diagnosis.—A pruriginous eruption of this kind over the bac and shoulders should always suggest a search for the pediculu vestimentorum. It is commonly found in the "gathers," unde the neck band of the shirt, or under the shelter of any edge pro jecting on the inner side; and it is recognised by its long ova shape, and its grey colour, with a central dark-red or black spot.

Treatment.—Ung. hydrarg. ammoniati or ung. staphysagria smeared over the skin, will kill the pediculi. The clothes, i which the eggs are certainly incubating, should be completel changed; and they must be baked if they are to be worn again.

PEDICULUS PUBIS.

The crab-louse is smaller than either of the other specie measuring from 1 to 1½ mm. long, and 1 to 1½ broad. It has a almost square body, and six long legs, with claws by which i clings firmly to the hairs of the part. It is not only found in th pubic hair, but is occasionally conveyed to the eyebrows, eyelashe whiskers, or beard. The eggs are attached to the hairs close t the skin. Itching leads to scratching, and an eczematous rash i the result. Removal of the hair quickly cures the trouble; o the pediculi may be killed by the means found effectual with othe species.

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